"CHANGES OF BRAINSTEM AUDITORY EVOKED POTENTIAL AND VISUAL EVOKED POTENTIAL IN VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACK PATIENTS"

Dissertation submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the

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(M.D. PHYSIOLOGY)

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THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERISTY

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APRIL – 2016

CERTIFICATE

This dissertation entitled **" CHANGES OF BRAINSTEM AUDITORY EVOKED POTENTIAL AND VISUAL EVOKED POTENTIAL IN VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACK PATIENTS "** is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulations for the award of M.D., Degree in physiology in the Examinations to be held during April 2016.

This Dissertation is a record of fresh work done by the candidate Dr. R.MOHAN, during the course of the study (2013-2016). This work was carried out by the candidate himself under my supervision.

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DECLARATION

I solemnly declare that the Dissertation titled **"CHANGES OF BRAINSTEM AUDITORY EVOKED POTENTIAL AND VISUAL EVOKED POTENTIAL IN VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACK PATIENTS"** is done by me at Thanjavur Medical College, Thanjavur

The Dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of requirements for the award of M.D. Degree (Branch V) in physiology

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CONTENTS

ABSTRACT

The Aim of the study is to compare the changes in Brainstem Auditory Evoked Potential and Visual Evoked Potential in VertebroBasilar Transient Ischemic Attack patients to the normal subject.

For this study, Auditory Evoked Potential and visual evoked potential were recorded in 40 patients with Vertebrobasilar Transient Ischemic Attack and 40 normal control group. This study was conducted at Research Laboratory , Department of Physiology,Thanjavur Medical College, Thanjavur. The Study group was from the Thanjavur Medical College & Hospital, Thanjavur.

Exclusion criteria were mainly Stroke, other Neurological diseases or Hearing loss. Informed written consent from the patients of Thanjavur Medical College & Hospital and control group was obtained.

In Brainstem Auditory Evoked Potential ,the Absolute Latency of Wave V was prolonged and amplitude reduction was observed in Wave V of Vertebrobasilar Transient Ischemic Attack patient and was found to be statistically significant.

In Visual Evoked Potential , no changes were observed in the Absolute Latency and amplitude of Vertebrobasilar Transient Ischemic Attack patient and was found to be statistically insignificant.

INTRODUCTION

Transient Ischemic Attack describes the symptoms of nervous system due to ischemic origin that lasts for less than 24 hours. Indeed most of the attacks last only few minutes to an hour. [1]

This transient attack is based on more than one mechanism. If any stenosis is present in the vertebrobasilar system, Transient ischemia will be caused by reduction of blood distally. The Transient ischaemia is mainly because of inadequate collateral bloodflow. VB TIA may have a hemodynamic basis which includes transient hypotension or cardiac arrhythmia. [1]

In clinical medicine Ischemia is the common form of cell injury which results from hypoxia that is induced by reduction in blood flow mostly it is due to mechanical obstruction of artery.[2]

So in ischemic tissues not only the aerobic metabolism is compromised, but also the energy generation by anaerobic metabolism stops having all glycolytic substances are exhausted. The accumulated metabolites will lead to inhibition of glycolysis. The biochemically altered reactions occur following ischemia, which will be summarized here. When the oxygen tension falls inside the cell, there will be loss of oxidative phosphorylation and the ATP generation will be decreased. This ATP depletion leads to sodium pump failure causes potassium efflux sodium and water efflux and eventually cell swelling. [2]

Influx of calcium also occurs which have many detrimental effects. Progressive loss of the glycogen, and reduction in protein synthesis occur. (2)

Attacks may be due to embolism from the heart or atherosclerotic plaque from the proximal large vessel. Because of the responsiveness of the TIA to calcium channel blockers, it suggests that the etiology is due to vasospasm. Lipohyalinosis and atherosclerotic plaques are caused by long standing hypertension ,diabetes or other vascular risk factors. (1)

It also has been associated with anemia,hyperviscosity,polycythemia, thrombocytosis, bacterial endocarditis,cerebral venous thrombosis,and it may clear with correction of the underlying disorder. (1)

TRANSIENT ISCHEMIC ATTACK:

It is an indicator of stroke. Twenty to twenty five% of the Ischemic stroke is due to a defect in the vertebrobasilar arterial Circulation.(3)

It is a temporary Neurological deficit of sudden onset. The onset of symptoms in TIA is sudden and it reaches its maximum intensity immediately. If it had not follow a complete clinical recovery, then it would not be a TIA.

Following are the symptoms that confirm the typical TIA in the posterior Circulation System. (3)

1. Bilateral dysfunction of the motor or sensory system.

2 .Either Complete loss of vision or partial loss of vision in the homonymous field of both eyes.

3. Vertigo, Diplopia, Dysphagia, Dysarthria and Ataxia.

To consider this as a TIA, the above symptoms should be present as an isolated or two or more combination of the symptoms.

Isolated presence of the above symptoms won't be consider as TIA.[3]

Changes of cerebral blood flow in VB TIA patients had studied by Drake ME et al on the basis of brainstem auditory evoked potential and visual evoked potential by P Benna et al. [4,5]

AIM AND OBJECTIVES

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This study was undertaken to compare the electrophysiological parameters between VB TIA patients and the control.

To evaluate the changes in blood flow in VB TIA patients by doing the electrophysiological parameters like brainstem auditory evoked potential and visual evoked potential.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

CEREBROVASCULAR ANATOMY:

VERTEBROBASILAR ARTERY:

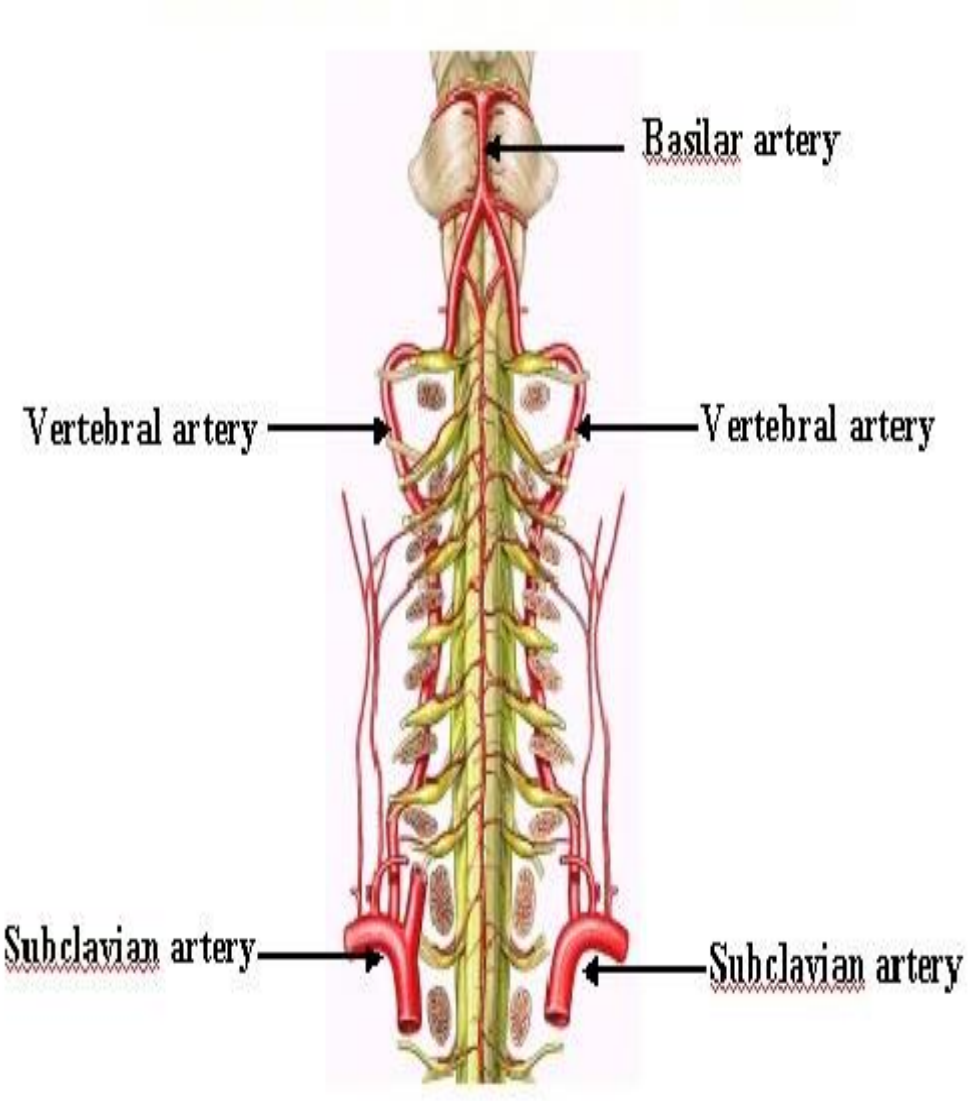


FIGURE:1

The brain receives blood supply from two pairs of arteries ,the right and left internal carotid arteries .Two vertebral arteries combines to form basilar artery . The basilar artery supply brainstem and posterior portion of cerebral hemisphere . This artery and the two internal carotid arteries unite to form the circle of Willis. [7]

CIRCLE OF WILLIS:

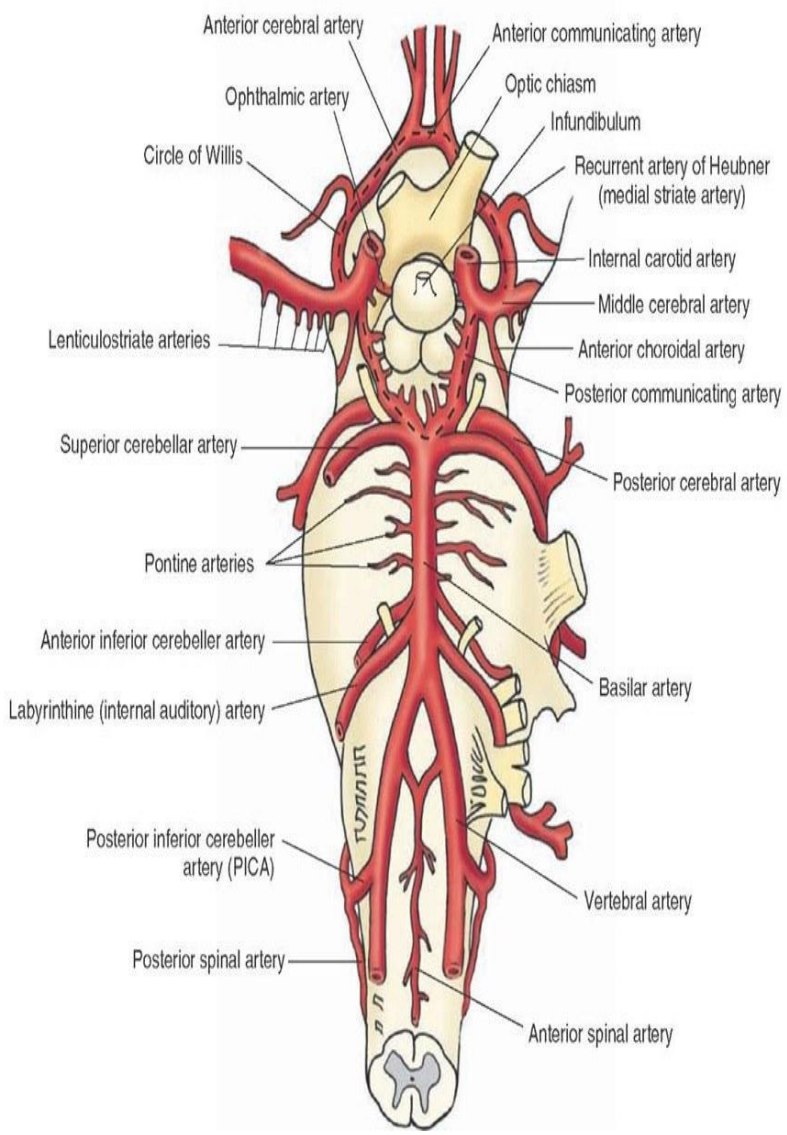


FIGURE:2

SUBCLAVIAN ARTERY:

The two vertebral arteries arise from the subclavian artery just proximal to the thyrocervical trunk in the neck. This subclavian artery also gives the following branches . They are internal thoracic artery, thyrocervicaltrunk,and costocervical trunk. [7]

COURSE IN CERVICAL VERTEBRAE:

It ascends and pass through the foramina of the transverse processes of the cervical vertebrae and enter into the skull via the foramen magnum. There will be a variations in the level of entry of the vertebral artery into the foramina of cervical vertebrae. This left subclavian artery arises directly from the aorta while the left subclavian artery arises from the Brachio -cephalic trunk. [7]

COURSES IN THE BRAIN:

Then it course along the anterolateral surface of the medulla where it gives branches that supply the medulla and the inferior aspect of the cerebellum before it forms the basilar artery at the pontomedullary junction. The basilar artery occupy the anterior surface of the brainstem and supplies the pons ,cerebellum and midbrain. Then it bifurcates into two posterior cerebral arteries which supply the postero-lateral aspect of midbrain,thalamus and occipital lobes.[7]

CORTICAL VASCULAR TERRITORIES:

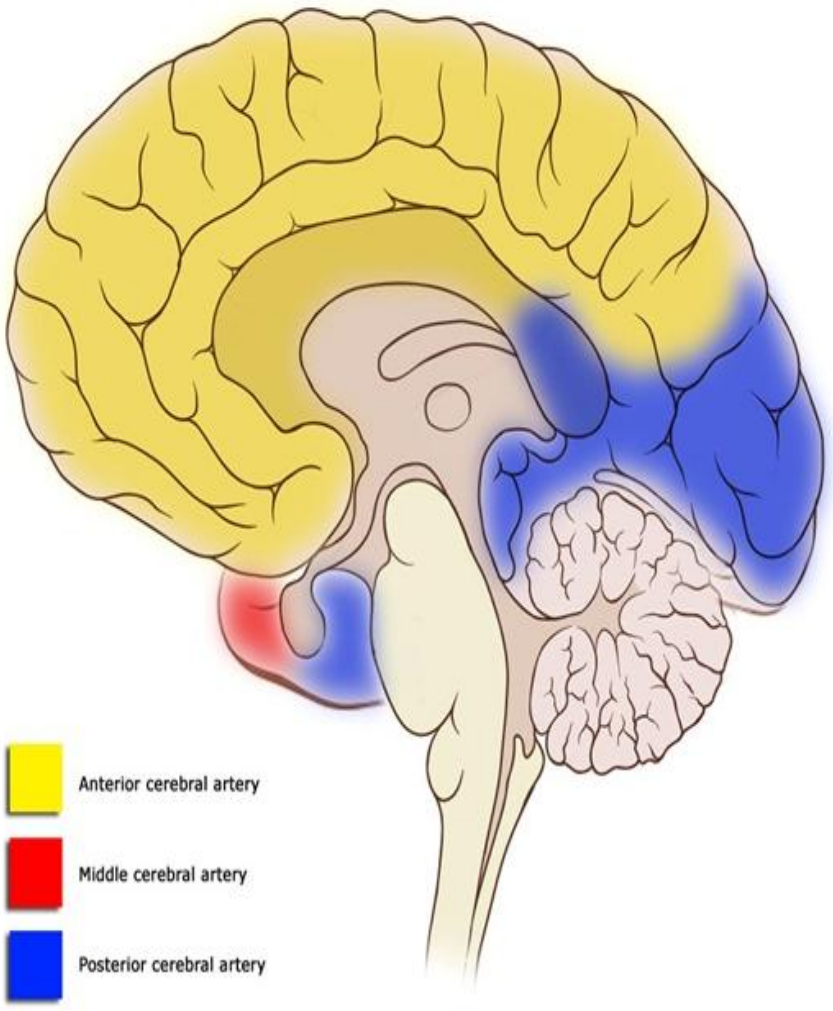


FIGURE:3

CAUSES OF CEREBRAL ISCHEMIA: [6]

VASCULAR DISORDER:

Atherosclerosis

Migraine

Connective tissue disorders

Drugs (Sympathomimetics)

Infective arteritis

Sickle cell anemia vasculopathy

Vascular dissection

Vascular trauma

Venous thrombosis

Vasculitis

Vasospasm

SYSTEMIC EMBOLISM:

Cardiac thrombus (Atrial Fibrillation, atrial myxoma, Dilated cardiomyopathy,Myocardial Infarction,Ventricular Aneurysm)

Fat Embolism.

Infective endocarditis

Valvular Heart disease

HAEMATOLOGICAL DISORDERS:

Hypercoagulable states (Disseminated Intravascular Coagulation ,Paroxysmal Nocturnal Hemoglobinuria ,Thrombotic Thrombocytopenic Purpura)

Hyperviscosity syndromes (PolyCythemiaVera , Myeloproliferative disorders,thrombocytosis)

OTHERS:

Nephritic Syndrome

Oral contraceptive pills

The causes of cerebral ischemia will be divided mainly into two categories. The one that cause Global ischemia and the others that cause ischemia of the focal area.Focal ischemia results from a local disturbances in the vascular system. The aetiology of the focal ischemia will be divided into three categories. [6]

Primary diseases of the cerebral vasculature, embolism arising from the extracerebral areas and disorders of the haematological system.Vascular compromise is the ultimate common pathway that results in inadequate supply of blood to a specific region in the brain, regardless of the aetiology. [6]

The alteration in the integrity of the endothelial wall initiate the formation of thrombus and it lead to disruption in the normal laminar blood flow. These changes will activate the coagulation cascade and subsequent thrombus production. The primary regulator of the thrombosis is thrombin, which activates the aggregation of the platelet and the formation of fibrin from the cleavage of the fibrinogen. [6]

This fibrin then forms the clot matrix by crosslinkage.This thrombus either can block the vessel wall at the injured site of the endothelium or embolize distally by shear stress and the action of antithrombotic intrinsic mechanism.

When the time progresses, these thromboemboli to the cerebral vasculature lyses spontaneously. Often this will occur too late to prevent the formation of permanent infarction.Atherosclerosis is the most common cause for the focal ischemia formation. It is formed by the multifactorial process and lead to the formation of atheroma. [6]

This atheroma contains cholesterol inflammatory cells, lipid laden macrophages,

abnormally proliferated endothelial smooth muscle cells and connective tissue forms within the vasculature. One of the key factors in the pathogenesis of atherosclerosis is the injury of endothelium which causes the formation of fatty streak and contributes to proliferation of smooth muscle cells. (6)

The source of thrombi which occlude the small cerebral blood vessels is the thrombus that is formed at the site of atheromatous plaque.

The factors which determines the formation of the atherosclerotic plaque will produce the symptoms of ischemia.

The basis for the atherosclerosis induced ischemia is the alteration in the local hemodynamic forces , luminal narrowing which exposes the thrombogenic contents of the necrotic core area.

Atherosclerosis may has a predilection for the vessel wall bifurcation and curves in vasculature. In the cerebrovascular system, the atherosclerosis occurs commonly at the beginning of internal carotid artery in neck area, distal part of vertebral artery, carotid siphon and in the basilar artery. The prevalence of atherosclerosis increases when the age advances. It affects younger men when compared to women at younger age.(6)

ATHEROSCLEROSIS RISK FACTORS: .[8]

- Old age
- Family history of thrombotic stroke
- Hypertension
- Diabetes mellitus
- Abnormal blood cholesterol level (low HDL, High LDL)
- Tobacco smoking

PATHOPHYSIOLOGY:

Based on the previous discussion ischemia will be defined as an impairing tissue perfusion with a view to reduction in blood flow relative to the metabolic demand. Vessel occlusion produces a massive reduction in blood flow to the central core of the tissue and a less reduction in the surrounding zone.

This surrounding tissue perfusion is maintained by the collateral circulation. There will be no change in brain metabolism as long as cerebral blood flow remains above 20 ml /100 gm / min .When the perfusion level reduces, the brain electrical activity fails and the neurological symptom arises. [6]

Since the oxygen supply becomes insufficient to maintain the normal cellular biochemical reaction, the high energy phosphate stores are quickly depleted.

Because of the meager supply of glucose to the tissue, anaerobic metabolism occurs which results in excessive lactate production.

By the time the CBF is more severely reduced to 10-12 ml/100 gm/min , the cell membrane integrity is lost and the depolarization leads to efflux of potassium ions and influx of calcium ions into the neurons. This increased extra cellular calcium results in increased energy consumption and the activation of the sodium-potassium ATP -ase pump in an attempt to restore intracellular potassium. Because the increased demand of the substrate cannot be compensated by increases in CBF, ischemia results.[6]

CLINICAL FEATURES:

Vertebrobasilar system ischemia will produce motor and sensory deficit. In addition to this, there will be a dysfunction of cranial nerve or cerebellum.

Small lesions of the brainstem produces unilateral isolated motor deficit. PCA involvement causes visual disturbances due to the ischemia of Occipital lobe.

Ischemia of the terminal branches of the vertebral basilar branches to the cerebellum produces ataxia. (6)

DEFINITION :

TRANSIENT ISCHEMIC ATTACK:

Abrupt loss of neurologic function occurred due to reduction in blood supply that persists for < 24 hours and resolves without residual disability. (6)

ISCHEMIA :

A constant supply of the oxygen and glucose will need for the brain's function which is supplied by cerebral blood vessel. Even though the brain weighing 1 to 2 percent of the body weight, it receives the resting cardiac output of fifteen percent. It receives about 20 percent of total oxygen consumption of our body. (6)

Because of the autoregulation of the cerebral vascular resistance, the cerebral blood flow remains constant over a broad range of blood pressure.

Brain is an aerobic organ so that the other metabolic substrate use is limiting. The transient ischemia is due to interruption in the normal cerebral blood flow.

This cessation of blood flow will result from a reduction of perfusion pressure .Impaired tissue function caused by reduction in blood supply and the metabolic demand.(3)

DIAGNOSTIC STUDIES:

Diagnostic studies include neuroimaging techniques , and CSF analysis are very much useful. The neuroimaging findings of the cerebral ischemia will significantly vary with time.

Within twenty four hours of the onset of the symptoms, the CT Scan will detect subtle signs of the infarction. The changes are blurring of the border between white and gray matter and loss of the sulci due to edema.

Magnetic resonance imaging of the brain is most sensitive to the ischemia. [6]

TREATMENT: .[6]

The aim of the treatment of the ischemic stroke is to limit the extent of the infarction and disability reduction.

It can be achieved by

I. Augmentation of fibrinolysis (thrombolytic drugs)

II. Salvation of neurons in the region of ischemic penumbra (agents with neuroprotective property)

III. To prevent the complication of stroke

The antiplatelet drugs and anticoagulant drugs role to prevent the progression of stroke is very much limited. The main goal of treatment in patients with ischemic stroke and the patients of TIA is to minimize the risk of subsequent stroke.

This is achieved through

1. To reduce the thrombotic potential by antiplatelet or the anticoagulant agents.
2. Modification of risk factors.

I . THROMBOLYTIC AGENTS :

Because the extent of brain injury which is induced by stroke is closely related to the severity and duration of ischemia, the clot lysing agents restore cerebrovascular perfusion eventually control the degree of injury. So that the outcome will improve by these agents.

In acute ischemic stroke ,intravenous streptokinase was tested. Because of the excessive haemorrhage, the trials were stopped.

Tissue plasminogen activator was trialled and it was accepted by the United states Food and drug administration. tPA was associated with excellent recovery, provided when it was administrated within first three hours of the onset of the symptoms. These thrombolytic agents shall be injected directly into the occluded microthrombi by infusion through microcatheter .

It had the extensive advantage of higher recanalization rate and safety improvement. The drug dose requirement in this situation is very much lower.

II. NEUROPROTECTIVE AGENTS:

The major goal of this therapy is to curtail the cellular death in the region of ischemic penumbra. So that the size of the infarct will be limited and the outcome will be improved.

Time is the important factor in efficient neuroprotective effect , since ischemia will irreversible within minutes to hours of the onset.

Anticoagulant and Antiplatelet therapy:

The role of aspirin in the treatment of acute stroke is limited. A randomized controlled trial was conducted with aspirin which revealed a reduction in early mortality among the patients. Heparin has no consistent benefit in stroke patients.

Even though the risk of recurrent stroke was reduced by heparin, it had the disadvantage to increase the extra cranial haemorrhage and haemorrhagic stroke.

III. PREVENTION OF THE COMPLICATIONS OF STROKE:

They are the major cause of morbidity and mortality among the stroke patients. The most common complication is hypertension.

Primary and secondary prevention of stroke and TIA:

General principles:

There are large number of strategies available to prevent the occurrence of stroke . They are medical therapies and surgical techniques as well as modification of life style.

Some of these techniques are applied in large extent on account of their low cost and with minimal risk. Others costs a lot of money and carry considerable risk but may be very useful for high risk patients. Identification and prevention of the modifiable risk factors is the best way of planning to minimize the burden of stroke. [6]

Hypertension is the most important risk factor. So all the hypertension must be treated. The Statin drugs are used to reduce the risk of stroke in patients even without the elevated level of cholesterol , which has confirmed by several trials.

SPARCL: [8]

(Stroke prevention by Aggressive reduction in cholesterol levels)

It showed usefulness in secondary reduction in stroke for patients with TIA. Atorvastatin 80 mg/ day was prescribed for this patient. Therefore statin must be prescribed in all patients with prior onset of ischemic stroke.

ANTIPLATELET AGENTS: .[8]

Atherothrombotic events like TIA and stroke can prevented by using antiaggregation

agents by inhibit the formation of platelet aggregates in the intraarterial system.

In the diseased artery the platelet aggregates formed ,which either occlude the artery or lead to distal embolization.

Agents to be used for this purpose are,

Aspirin

Clopidogrel

Aspirin + Extended release Dipyridamole

The most widely studied antiplatelet drug is aspirin.It activates the enzyme platelet cyclooxygenase, which inhibit the formation of Thromboxane A2 in platelets irreversibly. It is a prostaglandin produced from the pathway of cyclo-oxygenase with a property of powerful platelet aggregation.

This effect is permanent and its lasts for the life of the platelet. Aspirin also inhibit the production of Prostacyclin paradoxically. This Prostacyclin is an antiaggregating and vasodilating agents. When compared to aspirin, ticlopedine is more effective.

This study was undertaken to identify the changes of electrophysiological parameters in VB TIA patients when compared to normal subjects.

The electrophysiological parameters used in this study were VEP & BAEP.

The VEP represent the mass response of the cortical and subcortical areas.

If the entire visual system is intact, then only the normal cortical responses are obtained and the abnormal VEP will be produced by the disturbances anywhere in the visual system . Because of this property, localizing value of the VEP is limited.[9]

VEP is an electrical response recorded from the visual cortex in response to a change in visual stimuli such as multiple flashes of light (Flash visual evoked potential). It can identify the functional loss in the visual pathway from the visual cortex.

The visual stimuli may be unstructured as in a flashing light, or structured ,as in some form of pattern to the flash stimuli or the stimuli may be patterned, as in checkerboard presented on a video display unit. The essential feature is that while the pattern changes, the overall illumination remain the same. Black squares go white and white become black and this will occur alternatively. The rate of the dark square become lightening being the same as that of the darkening of the light squares.[10]

FLASH VISUAL EVOKED RESPONSE:

This is a most crude test and it indicates that light has been perceived. It is a fovea dominated response and is relatively unaffected by opacities in the cornea and the lens. It is therefore a useful test to grossly assess the intactness of the macula or the optic nerve. [11]

PATTERN REVERSAL VISUAL EVOKED RESPONSE :

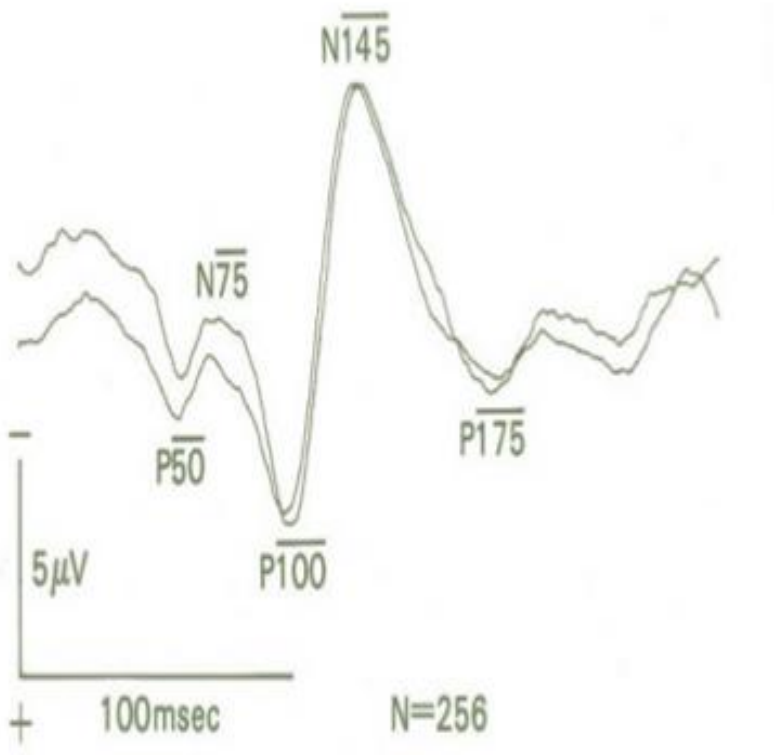
This depends on the form sense and may give a rough estimate of visual acuity. It is more of fovea specific response .the timing of responses are more reliable than the amplitude. The preferred stimulus for the visual evoked potential testing is a checkerboard pattern of black and white squares.[11]

NORMAL VEP FINDINGS:

The VEPs consists of a series of forms opposite polarity. The negative waves are denoted by N and positive waves by P ,which is followed by the approximate latency in ms. The commonly seen wave forms are N75,P100,and N145. The peak latency and peak to peak amplitudes of these waves are measured. [9]

VISUAL EVOKED POTENTIAL:

FIGURE:4



BASIS OF VEP ABNORMALITIES:

The VEP abnormalities may be latency prolongation, amplitude reduction and combined latency and amplitude abnormalities. The commonest cause of prolonged P100 latency is demyelination in the optic pathways where the amplitude of P100 remains normal. [9]

CLINICAL USES OF VEP:

The VEP study is a sensitive method for detecting the abnormalities in visual pathways especially anterior to the optic chiasma.It should be regarded as complementary to clinical examination and neuro-ophthalmological investigations.[9]

CHECKER BOARD PATTERN OF VEP:

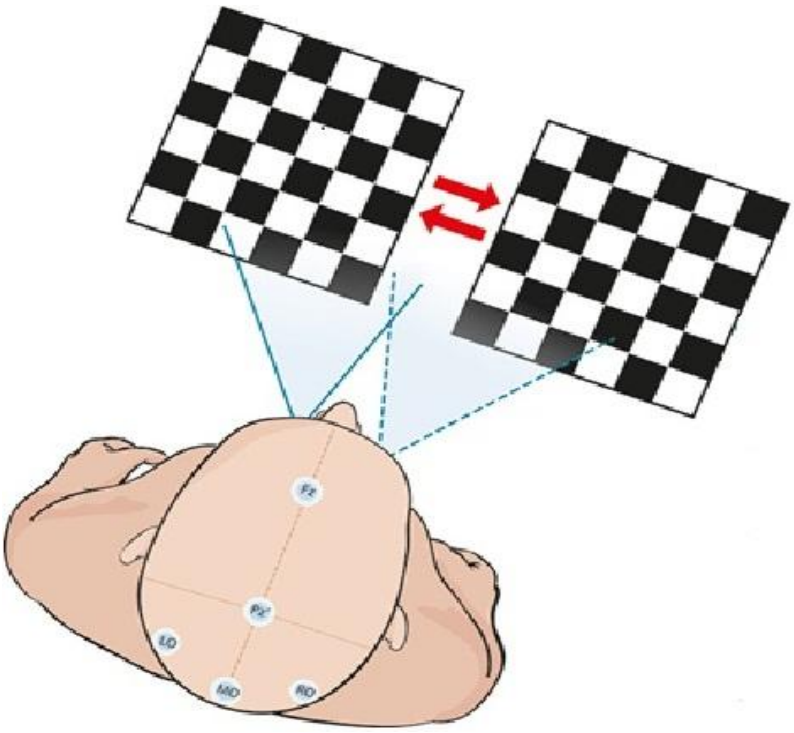
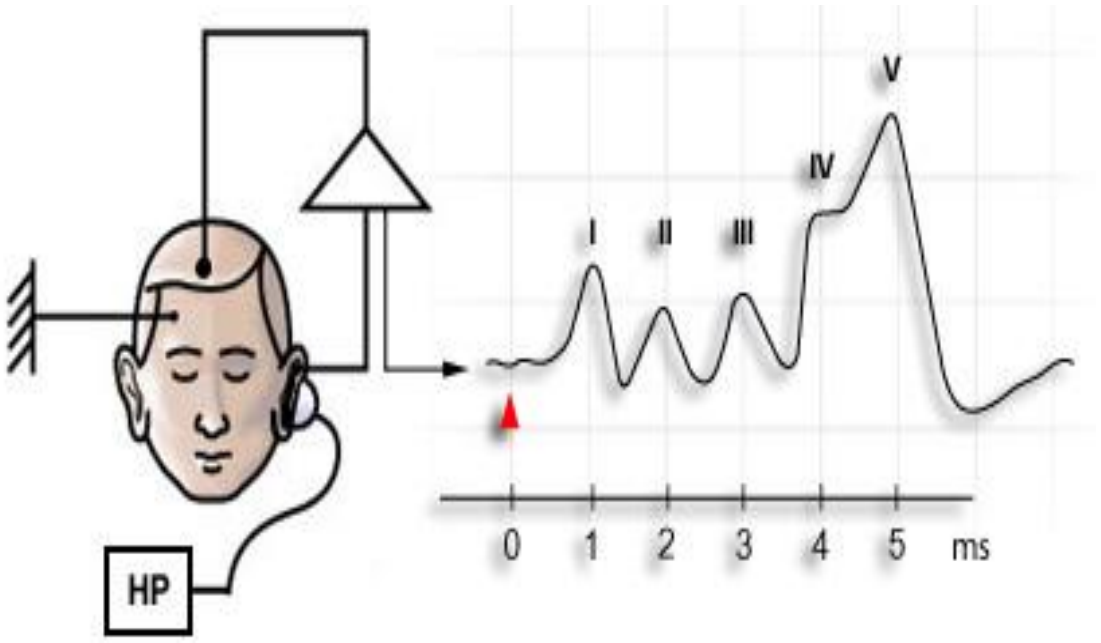


FIGURE:5



BRAINSTEM AUDITORY EVOKED POTENTIAL:

Brainstem auditory evoked potentials (BAEP) are the potentials recorded from the ear by placing the electrodes in the scalp in response to a brief auditory stimulation to assess the conduction through the auditory pathway upto midbrain.

The evoked potentials that appear following transduction of the acoustic stimulus by the ear cells create an electrical signal that is created through the auditory pathway to the brainstem and from there to the cerebral cortex. BAEPs comprise five or more waves within 10 ms of the stimulus. It may describe in terms of duration of onset of response. [12]

WAVE I – Cochlear nerve

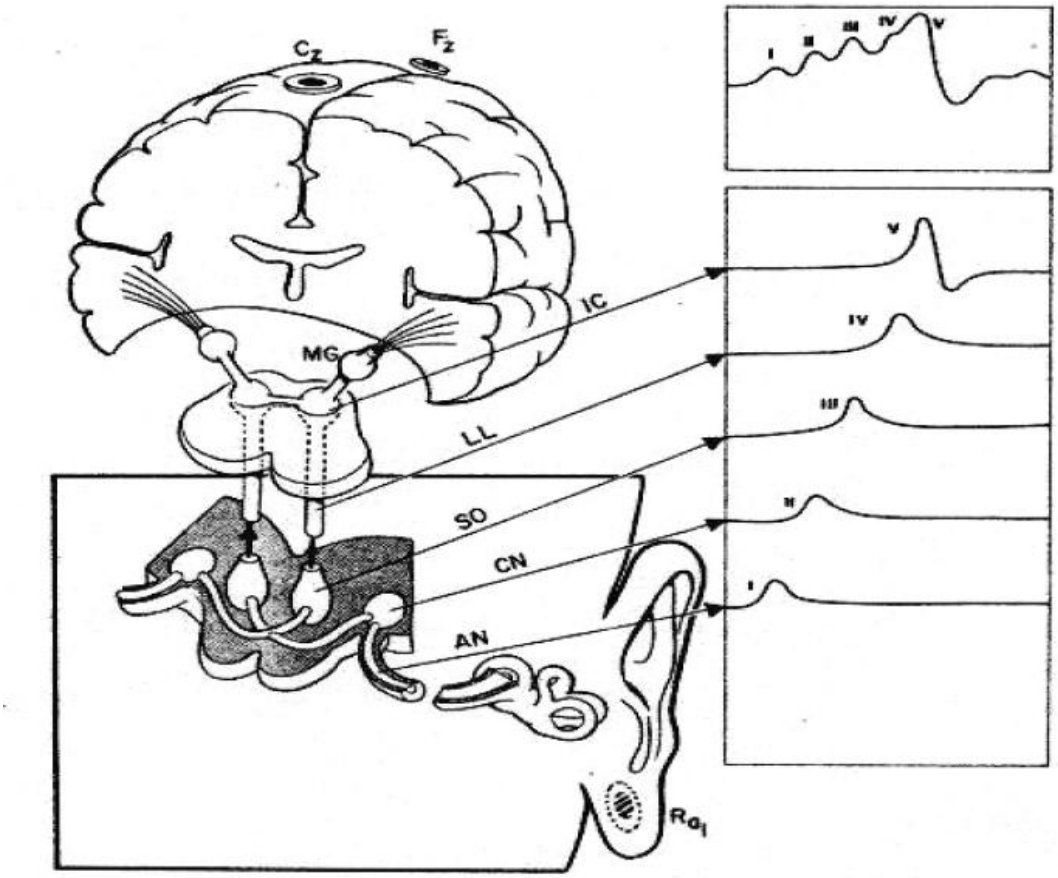
WAVE II – Cochlear nucleus

WAVE III – Superior Olivary Nucleus

WAVE IV – Lateral Lemniscus

WAVE V – Inferior Colliculus

FIGURE:7



AUDITORY PATHWAY:

In human being the sound reaches the brain through the ear. The frequency of sound that humans can hear ranges from 20 - 20,000 Hz.

Then it travels through the outer, middle, and inner ear. In the Organ of Corti the mechanical sound waves are converted into electric neural signal.

Then, the dendrites of primary auditory neurons communicate with these signal. Later they are bundled into cochlear nerve after which they join the vestibular nerve to form Vestibulo cochlear nerve. Information is transferred through cochlear nuclei reaches the Superior Olivary complex and finally through the Inferior Colliculus , Lateral Lemniscus to the thalamus. At the level of cochlear nucleus this sound waves cross to the opposite side. [13,14]

The Inferior Colliculus within the midbrain is divided into dorsal part nucleus and the central nucleus. The dorsal part receives both somatosensory and auditory input. The central nucleus is involved in auditory localization. Within the thalamus, there is an oval structure found within diencephalon which conveys sensory input to the Medial Geniculate Nucleus (MGN).As it act as a primary sensory area it represents a major relay station for auditory system. The Medial Geniculate Nucleus (MGN) consists of three subdivisions. Among which, the principal nucleus receives the auditory input. [13,14]

From MGN, information is relayed to the primary auditory cortex which is

otherwise called us A1.It is located on transverse Gyrus of Heschl situated within temporal lobe. This auditory cortical area allows the sensation of auditory characteristics like pitch.

This primary auditory cortex is composed of many functional columns Neurons found within same column process sounds of the same frequency. Further, they are tonotopically organized in similar to all the previous stages of auditory processing mentioned above. (15)

Auditory neurons are spatially arranged in an order– according to auditory frequencies they process. There is more evidence that its distinction into two different pathways with peculiar functions like in the visual cortex can be found in auditory cortex. (16)

VISUAL PATHWAY:

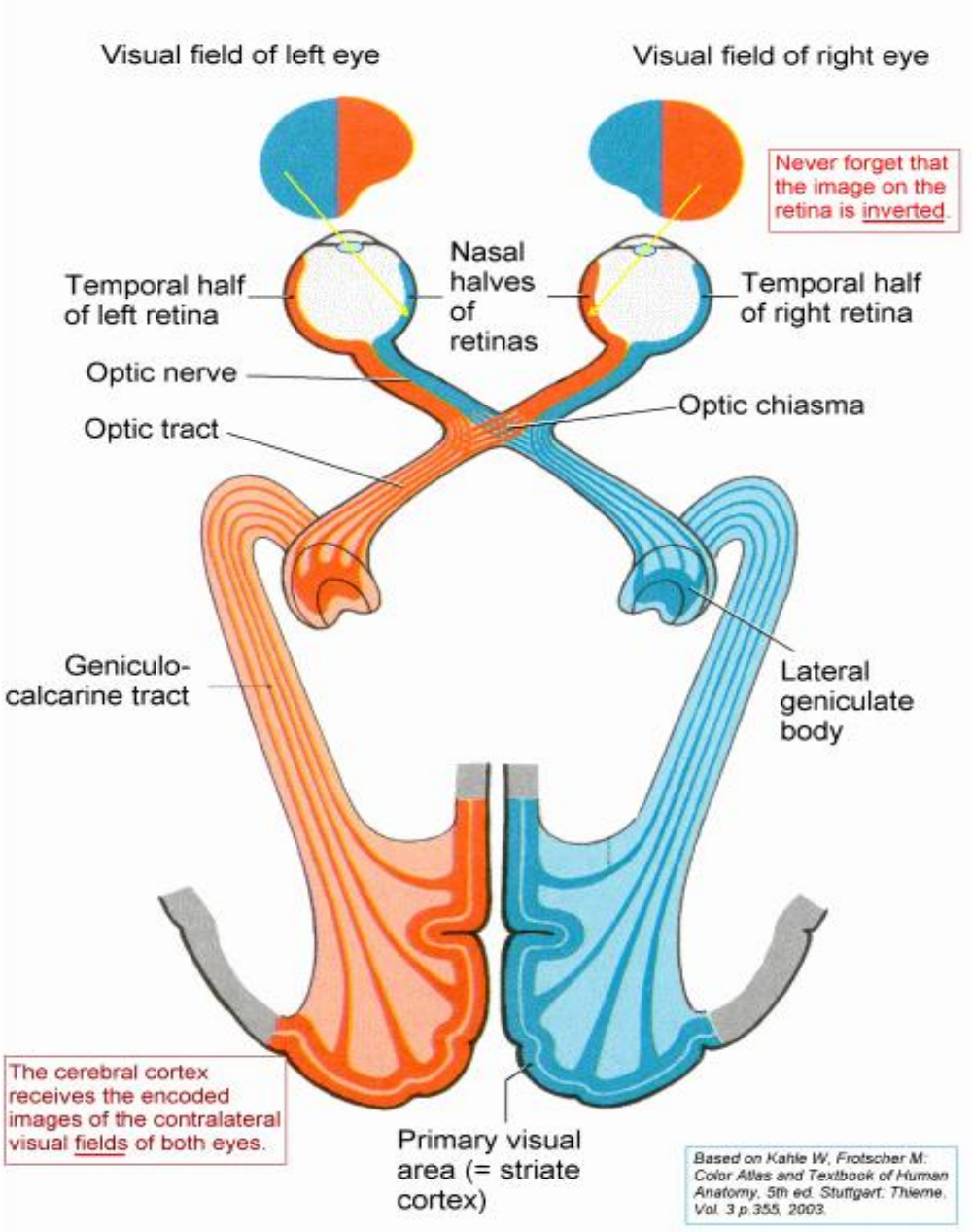


FIGURE: 8

VISUAL PROCESSING AND VISUAL PATHWAY:

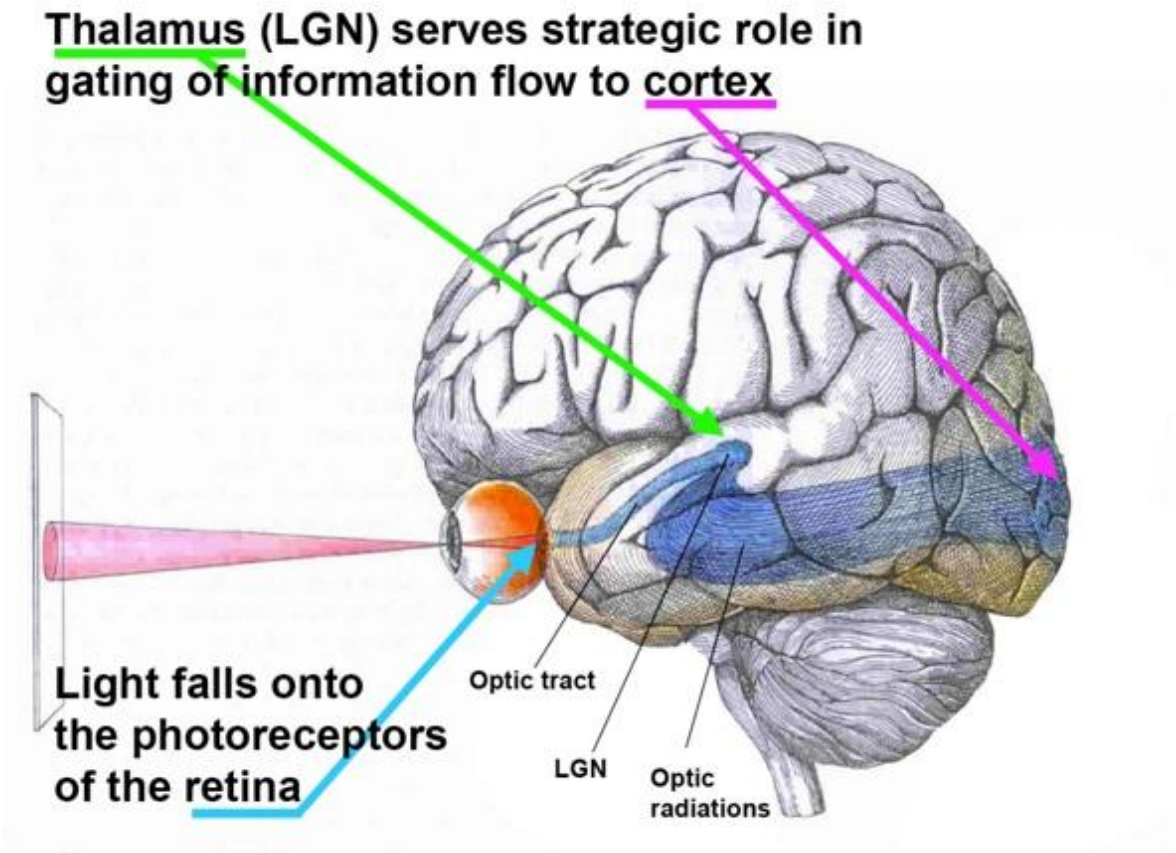


FIGURE: 9

The ganglion cell's axon of the retina passes caudally in the optic nerve fibre and optic tract which eventually end in the lateral Geniculate body. The fibers from each nasal half of retina crosses in the optic chiasm and leaves as optic tract whereas the temporal fibers will not decussate.[17]

In the lateral geniculate body, the nasal half of one retinal fibers and the other temporal half of the fibers synapse on the cells. The axons of these geniculate bodies form the geniculocalcarine tract.This geniculocalcarine tract passes to the occipital lobe of the cortex. Brodmann classify the visual cortex into 17,18 and 19. Brodmann's area 17is the primary visual cortex, Which is also known as V1.This primary visual cortex is located principally in the sides of the calcarine fissure . The organization of the primary visual cortex is summarized in the following.

Pretectal region of the midbrain also receives Some of the axons of ganglion cell.[17]

BAEPs are much more useful to study in assess the objective function of the auditory system and also noninvasively. It specifically assess the cochlea-auditory nerve – brainstem pathway resulting in an extensive development of recording of both ear by placing the electrodes over the scalp and the far field electric potentials.

EARLY AUDITORY EVOKED POTENTIALS:

Early auditory evoked potentials have also been reported to as short-latency auditory evoked potentials and corresponding to the responses recorded within the initial twelve millisecond after the given auditory stimulus. .[18]

MIDDLE LATENCY AUDITORY EVOKED POTENTIALS:

Middle latency Auditory evoked potentials are potentials occurring between

12 and 50 msec after an acoustic stimulation.. They can be recorded from transient or from high frequency stimuli. Middle latency Auditory evoked potential has been clinically applied in the assessment of hearing threshold in infants and children, the identification of dysfunction in central auditory pathways, and the evaluation of the central auditory pathways in candidates for cochlear implants. .[18]

LATE AUDITORY EVOKED POTENTIALS:

Evoked potentials occurring 50 msec after an acoustic stimulation are called slow or late auditory evoked potentials. These potentials can be subdivided into exogenous components. N1,P1 and P2 which are primarily dependent on characteristics of the

external stimulus ,and endogenous components such as P300,N400,CNV,

and the mismatch negativity, which are more dependent on internal cognitive processes. [18]

NORMAL BAEP FINDINGS:

The BAEP consists of five or more distinct wave forms recorded within 10 msec of the auditory stimulus and they are generated in different regions of the peripheral and central auditory pathways. Wave I is originated from the peripheral portion of the auditory system adjacent to the Cochlea.

Wave II originates from the cochlear nucleus, wave III from the superior olivary nucleus Wave IV from the Lateral lemniscus , and Wave V from inferior colliculi. The Absolute Latencies and Interpeak latencies I-III,III-V and ,I-V were measured. Amplitude of Wave I and Wave V were measured. [18]

INTERPRETATION OF BAEPS:

BAEP interpretation requires identification and measurement of waves I,III,and V , the measurement of I-V and I-III intervals. These values could be compared with the normal values of the patients.

First the absence of wave I with normal Wave V will probably reflects the technical problem in recording. Then absence of wave III is significant only when wave V is also missing or delayed. Finally BAEP s cannot be interpreted without considering the patients hearing status; conductive hearing loss and cochlear pathology may profoundly affect BAEP wave late latency and amplitude. [19]

FINDINGS OF EVOKED POTENTIALS IN VB TIA PATIENTS:

Drake ME et al Conducted a study of BAEP changes in vertebrobasilar transient ischemic attacks patients . The study was recorded after resolution of symptoms ,

Some of the patient in this study still had resolving symptoms or signs. The findings

in TIA patients were longer I-III, III-V, and I-V interpeak latencies , but these were not significant when compared to controls. Absolute latency of Wave V was significantly longer and amplitude of wave V is lower in TIA patients.[4]

P Benna et al conducted a study of brainstem evoked auditory response and visual evoked potentials of pattern reversal in patients suffering from vertebrobasilar TIA

with a view to obtain the functions of cortical-subcortical areas. BAEPs alterations occur in patients with vertebrobasilar TIA .The parameters of VEPs are normal in both group.[5]

Stewart A et al conducted Brain-stem auditory evoked response (BAER) at about few days to two weeks after a vertebrobasilar transient ischemic attack (VB TIA) . Initially it showed absence of all waveforms, prolongation of I –III , III –V and I-V inter-peak latencies and reduction of amplitude . Some of the patients showed reversal of latencies and amplitude to normal. Normalization of the waves occurred 6 to 24 days after the VB TIA. The result of this studies are different from other reported studies. [25]

Factor S A et al studied Brain-stem auditory evoked response in vertebrobasilar transient ischemic attack (VB TIA) patients . In the beginning , all showed, prolonged interpeak latencies, absence of waveforms and reduction in amplitude . 5 out of 6 patients showed reversal of the changes to normal.In rest of the patients it returned to near normal. 6 to 24 days after the VB TIA attack the results came to normal. These results are very much different from other reported studies.[20]

Meier U et al Studied the value of brain stem auditory evoked potentials in vertebrobasilar circulation to diagnose the circulatory disorders. The evoked brain-stem potential in patients of vertebrobasilar insufficiency showed variation in diagnostic evaluation in the literature. Their examination revealed an advantage in subdividing the BAEP changes into normal , slight and pronounced, within this group of patients of the vertebrobasilar region blood supply disorders. Depending upon the extent of the damage, the changes of BAEP varied .[21]

Ferbert A et al studied the Evoked potentials in diagnosis of brain stem ischemia. They presented the impact of evoked potentials in diagnosis of ischemic lesions of the brainstem. They found Prolonged interpeak latency of Wave I-III of Brainstem auditory evoked potentials in patients with caudal occlusions, whereas visual evoked potentials were normal in most of the cases.

The Brainstem Auditory Evoked potential changes are found in patients with basilar artery thrombosis and primary hemorrhage of the pontine area indicating the location of the lesion. Further they are of prognostic value. Depending on the location of the infarction , the evoked potential changes are found in basilar artery occlusive stroke.[22]

Thorwirth V et al studied the Brain stem Auditory evoked potentials and visual pattern evoked in transient ischemic attacks. Auditory-evoked brainstem potentials, visual pattern-evoked and somatosensory-evoked potentials in transitory ischemic attacks (TIA). They found that without any diagnostic risk , the combination of visual evoked potentials and auditory brainstem-evoked can detect the functional lesions in regions of cerebrum with different blood flow .

Their results showed that there was a delay in the peak of Wave III of the BAEP in TIA in patients and possibly of specific importance in brainstem lesion.[23]

Zhang X. J et al studied the Clinical Value of Brainstem Auditory Evoked Response in the Diagnosis of Vertebrobasilar Ischemia. They found an abnormal value of BAEP in the patients of vertebrobasilar ischemia (VBI).

BAEP is an easy and effective means for the examination of VBI patients and providing objective evidence to diagnose Vertebro basilar ischemia which may be useful for future application.[24]

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was conducted in the department of physiology, Thanjavur medical college & Hospital, Thanjavur. The study was case control type and conducted between January 2015 and July 2015. The patients were selected from the department of Internal medicine.

In the 40 VB TIA Patients , 24 males and 16 females were selected in the age group of 50-70. VB TIA was confirmed by the Neuroimaging technique MRI . History of the patient was asked and complete examination including general and systemic examination was done. In the control group 23 males, 17 females of age group 50-70 years were selected.

Exclusion criteria :

Neurological diseases

Hearing defects

Stroke

Hypertension

Diabetes mellitus

Meniere 's disease

Migraine

The Procedure was explained to all the persons who were participated . Informed written consent was taken from both the controls and subjects . The ethical committee was given an approval to this study.

The following electrophysiological parameters were studied:

- Visual Evoked Potential (VEP)
- Brainstem Auditory Evoked Potential (BAEP)

All the parameters were recorded using four channel Digital Polygraph. Digital Intex colour Monitor, 17" Model no : IT – 173 SB.

Medicaid neuroperfect plus Instrument was used in this study .

METHOD OF RECORDING VEP,BAEP:

Electrodes were placed using 10-20 electrode placement system.[26]

VISUAL EVOKED POTENTIAL:

Pre test instructions:[9]

1. The subject was explained about the procedure of this test .
2. Informed consent were obtained.
3. The subject is instructed to avoid applying hair oil after their last hair Wash.
4. The subject can use their Optical lenses/Glasses during the test.

5 .The subject is instructed not to use any mydriatics or miotics during the test.

6. Complete Ophthalmological examination was carried out to determine the visual acuity , field of vision.

7.The Room should be quite and comfortable.

RECORDING OF VEP:

Settings for VEP:

SETTINGS	UNITS
Sweep	20 msec
Sensitivity	10 µV
Low cut	2 Hz
High cut	200 Hz
Pulse	1/sec
Pulse Width	0.1 msec
Notch	On
Recordings	100 average was recorded using Checker Board pattern stimulus

PROCEDURE:

The visual evoked potential which was tested by pattern reversal was measured separately for both eyes by the following steps,

1.The skin is prepared by degreasing.

2. The Recording electrode is placed at Oz using Ten 20 CONDUCTIVE Neurodiagnostic electrode Paste as per 10-20 international system of EEG electrode placement. [26]

3.Reference electrode is placed at FPz.

4. The Ground electrode is placed at Cz.

5. The procedure is conducted in dark room with subject sitting at a distance of 1 meter from the VEP screen which is showing pattern reversal stimuli in Checker board pattern with reversal rate 2/sec contrast 50-80% check size 28-32 of arc and number of trails is 100.

6.Values are marked in the obtained waveform.

BRAINSTEM AUDITORY EVOKED POTENTIAL:

Pre test instructions: [9]

1.The Subject was explained about the procedure of the test.

2.Informed consent was obtained from the subjects.

3. The subject is asked to avoid applying hair spray or hair oil after the last hair wash.

4.Examination of the external ear was done & if any wax was found it was removed.

4.Tunning fork test were carried out examples are Rinne's, Weber's & Absolute bone conduction test.

5.Subject is made to relax completely.

6.Room should be quite and comfortable.

RECORDING OF BAEP:

Instrument settings for BAEP:

SETTINGS	BAEP
Sweep	5 msec
Sensitivity	10µV
Low cut	100 Hz
High cut	10 Hz
Pulse	11/sec
Pulse Width	0.1 msec
Notch	ON
Decibels	60 Db
Recordings	100 average was recorded using click sound as a stimuli

PROCEDURE:

1.The skin is prepared by abrading and degreasing

2.The electrode is placed at

Channel 1 = Cz-Ai (ipsilateral ear)

Channel 2 = Cz – Ac (contralateral ear)

Ground electrode is placed at 20% from the nasion Fz

3. Head phones are placed on the ears for delivery of the auditory stimuli. Clicks are delivered at the rate of 8-10/sec.Intensity is to set at 60 db. About 100 Averages are taken.

4. Wave I to V are marked in the obtained wave form. Wave I is the first major upgoing peak following a stimuli.

5.Wave V is appear at approximately 6 ms and is often combined with wave IV into a single complex waveform. Wave III is the major peak between wave I and V. Wave II is typically the first major upward deflection in the Cz – Az waveform as Wave I is markedly attenuated or absent.

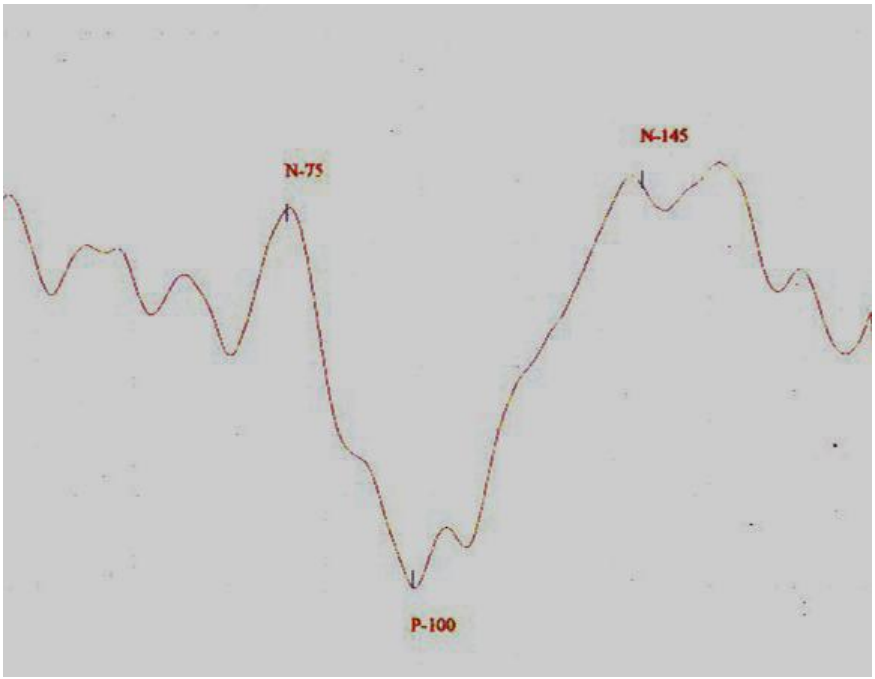
6. From the above wave form interpeak latency I-III, III-V , and I – V is obtained.

Statistical method:

Electrophysiological parameters were analysed by using statistical package SPSS version 18 and statistical analysis was done by student " t " test.

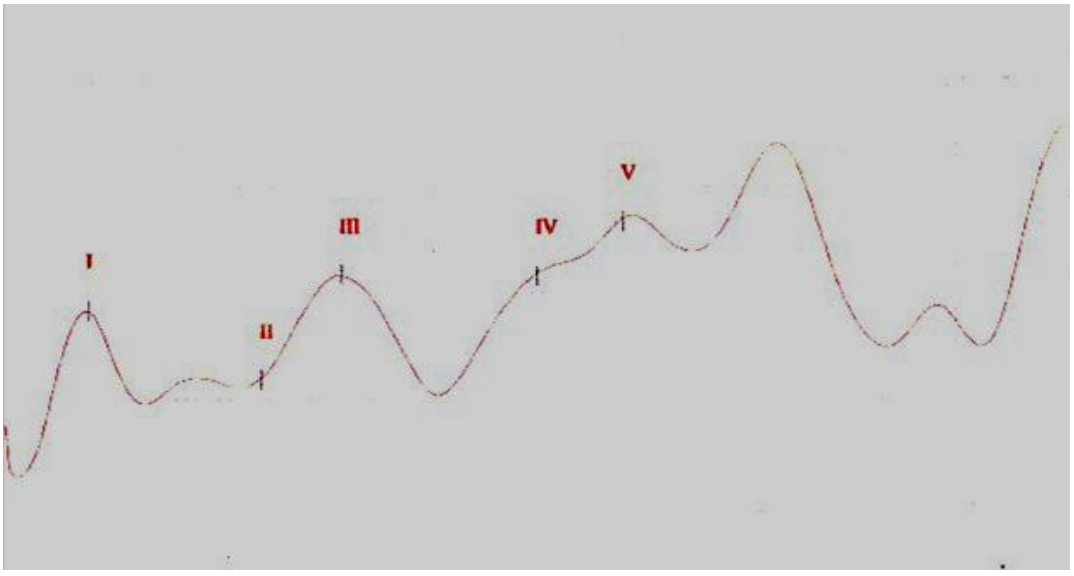
VEP RECORDING

VEP WAVEFORM



BAEP RECORDING

BAEP WAVEFORM



RESULTS

RESULTS:

In this study 80 subjects were included. 40 VB TIA patients were in study group and rest of the 40 normal subjects were in control group.

In this study , 40 VB TIA study group were in the age of 50-70 years, mean 58.725 ± 4.272 and control groups were in the age group of 50-70 years, mean 58.975 ± 4.457.

The two groups differ significantly in BAEP. P value was derived from data analysis using statistical package SPSS version and statistical analysis was done by student ‘t’ test. The statistical significance was considered when P value < 0.05 .

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DESCRIPTIVE STATISTICS

TABLE 1: BAEP LATENCY Findings –CONTROL (n = 40)

LATENCY (ms)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
WAVE I	RIGHT	1.6	1.9	1.71575	0.075035
	LEFT	1.6	1.85	1.7125	0.064639
WAVE III	RIGHT	3.55	4.08	3.776	0.105704
	LEFT	3.45	4	3.76275	0.101829
WAVE V	RIGHT	5.42	5.85	5.703	0.09962
	LEFT	5.42	6.02	5.74575	0.133607
WAVE I-III	RIGHT	1.85	2.46	2.06025	0.126054
	LEFT	1.6	2.3	2.05025	0.128731
WAVE III-V	RIGHT	1.57	2.25	1.927	0.154691
	LEFT	1.6	2.5	1.983	0.173104
WAVE I-V	RIGHT	3.7	4.2	3.98725	0.106962
	LEFT	3.67	4.27	4.03325	0.157046

TABLE 2 : BAEP LATENCY Findings –SUBJECT (n = 40)

LATENCY (ms)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
WAVE I	RIGHT	1.62	1.87	1.7295	0.057197
	LEFT	1.62	1.88	1.7275	0.076149
WAVE III	RIGHT	3.6	4.02	3.77925	0.103686
	LEFT	3.62	3.98	3.78125	0.101633
WAVE V	RIGHT	5.42	6.08	5.74125	0.13915
	LEFT	5.52	5.98	5.7985	0.114635
WAVE I-III	RIGHT	1.81	2.3	2.04975	0.11652
	LEFT	1.85	2.33	2.06025	0.127192
WAVE III-V	RIGHT	1.58	2.4	1.962	0.187195
	LEFT	1.66	2.3	2.01725	0.162969
WAVE I-V	RIGHT	3.73	4.39	4.01175	0.137204
	LEFT	3.8	4.22	4.071	0.118013

TABLE 3 : BAEP Findings – P value

LATENCY (ms)	SIDE	CONTROL	SUBJECT	P-VALUE
WAVE- I	LEFT	1.7125	1.7275	0.18
	RIGHT	1.71575	1.7295	0.13
WAVE-III	LEFT	3.76275	3.78125	0.09
	RIGHT	3.776	3.77925	0.39
WAVE-V	LEFT	5.74575	5.7985	0.02 **
	RIGHT	5.703	5.74125	0.04 **
WAVE I-III	LEFT	2.05025	2.05375	0.44
	RIGHT	2.06025	2.04975	0.25
WAVE III-V	LEFT	1.983	2.01725	0.13
	RIGHT	1.927	1.962	0.11
WAVE I-V	LEFT	4.03325	4.071	0.09
	RIGHT	3.98725	4.01175	0.16

TABLE 4 :BAEP Amplitude Findings – SUBJECT

AMPLITUDE (μV)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
WAVE I	RIGHT	3.02	22.15	9.972	5.401
	LEFT	3.58	13.42	8.3375	2.630
WAVE V	RIGHT	8.01	31.64	17.146	6.971
	LEFT	8.28	37.84	17.449	6.882

TABLE 5 :BAEP Findings – CONTROL

AMPLITUDE (μV)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
WAVE I	RIGHT	3.31	15.64	9.482	3.61
	LEFT	4.12	18.26	8.978	3.619
WAVE V	RIGHT	7.05	42.31	20.498	9.359
	LEFT	8.75	36.58	19.95	5.670

TABLE 6 :BAEP Findings – 'P' Value

AMPLITUDE(μV)	SIDE	SUBJECT	CONTROL	P VALUE
WAVE I	RIGHT	9.972	9.482	0.32
	LEFT	8.337	8.978	0.19
WAVE V	RIGHT	17.146	20.498	0.05 **
	LEFT	17.449	19.95	0.04 **

TABLE 7 :VISUAL EVOKED POTENTIAL – CONTROL (n=40)

LATENCY(ms)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
N75	RIGHT	69	78.5	73.2625	2.081628
	LEFT	69.5	80	73.2375	2.345174
P100	RIGHT	95	108	100.512	2.8227
	LEFT	91.5	117.5	101.687	4.8047
N145	RIGHT	139	152	145.487	2.9515
	LEFT	138	151	143.637	3.2679

TABLE 8 :VISUAL EVOKED POTENTIAL – SUBJECT (n = 40)

LATENCY(ms)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
N75	RIGHT	70	77	73.037	1.677
	LEFT	69.5	78	72.95	2.0933
P100	RIGHT	97	105	100.775	1.822
	LEFT	95	115	101.812	3.553
N145	RIGHT	138.5	152	145.475	2.832
	LEFT	137.5	149.5	144.1	2.539

TABLE 9 : VISUAL EVOKED POTENTIAL – P VALUE

LATENCY(ms)	SIDE	SUBJECT	CONTROL	P VALUE
N75	RIGHT	73.037	73.2625	0.21
	LEFT	72.95	73.2375	0.14
P100	RIGHT	100.775	100.512	0.22
	LEFT	101.812	101.687	0.38
N145	RIGHT	145.475	145.487	0.44
	LEFT	144.1	143.637	0.07

TABLE 10 : VISUAL EVOKED POTENTIAL - CONTROL

AMPLITUDE (µV)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
P 100	RIGHT	3.02	22.15	11.433	1.919383
	LEFT	3.58	13.42	11.358	2.221044

TABLE 11 : VISUAL EVOKED POTENTIAL - SUBJECT

AMPLITUDE(μV)	SIDE	MINIMUM	MAXIMUM	MEAN	STANDARD DEVIATION
P 100	RIGHT	3.02	22.15	11.477	1.834364
	LEFT	3.58	13.42	11.345	2.142257

TABLE 12 : VISUAL EVOKED POTENTIAL - P VALUE

AMPLITUDE (μV)	SIDE	CONTROL	SUBJECT	P VALUE
P 100	RIGHT	11.433	11.477	0.12
	LEFT	11.358	11.345	0.4

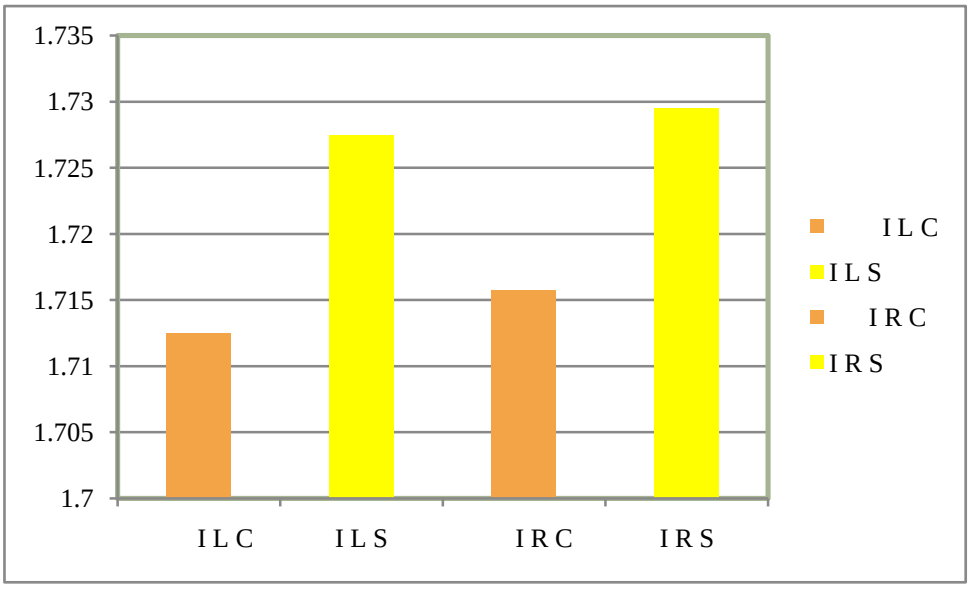

FIGURE:10

The Absolute Latency of Wave I of left side in VB TIA patients show the mean value of 1.7275±0.076149 and in the control group it is 1.7125±0.064639 with a P value of 0.18 showing the difference insignificant.

The Absolute Latency of Wave I of right side inVB TIA patients show the mean value of 1.7295±0.057197 and in the control group it is 1.71575±0.075035 with a P value of 0.13 showing the difference insignificant.

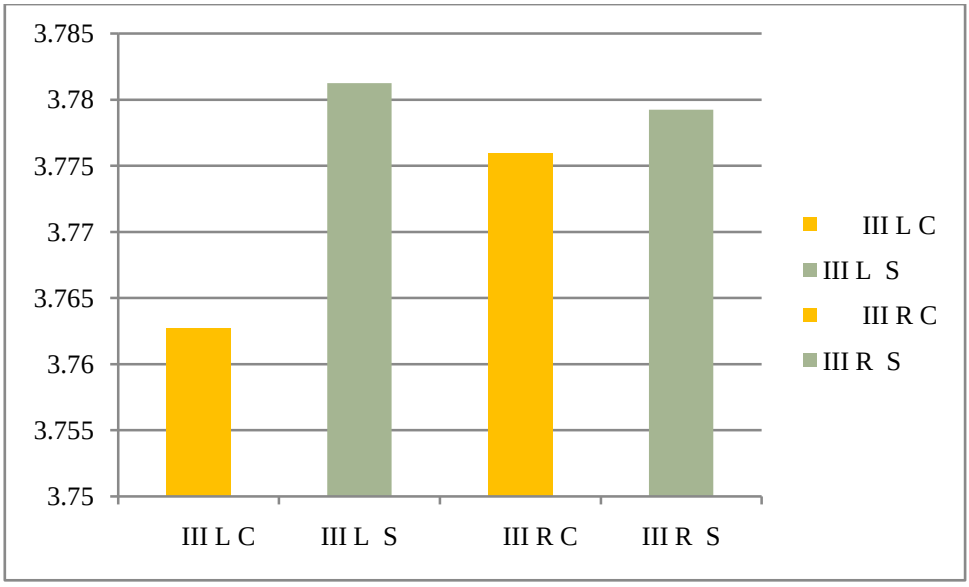

FIGURE:11

The Absolute Latency of Wave III of left side in VB TIA patients show the mean value of 3.78125±0.101633 and in the control group it is 3.76275±0.101829 with a P value of 0.09 showing the difference insignificant.

The Absolute Latency of Wave III of right side in VB TIA patients show the mean value of 3.77925±0.103686 and in the control group it is 3.776±0.105704 with a P value of 0.39 showing the difference insignificant.

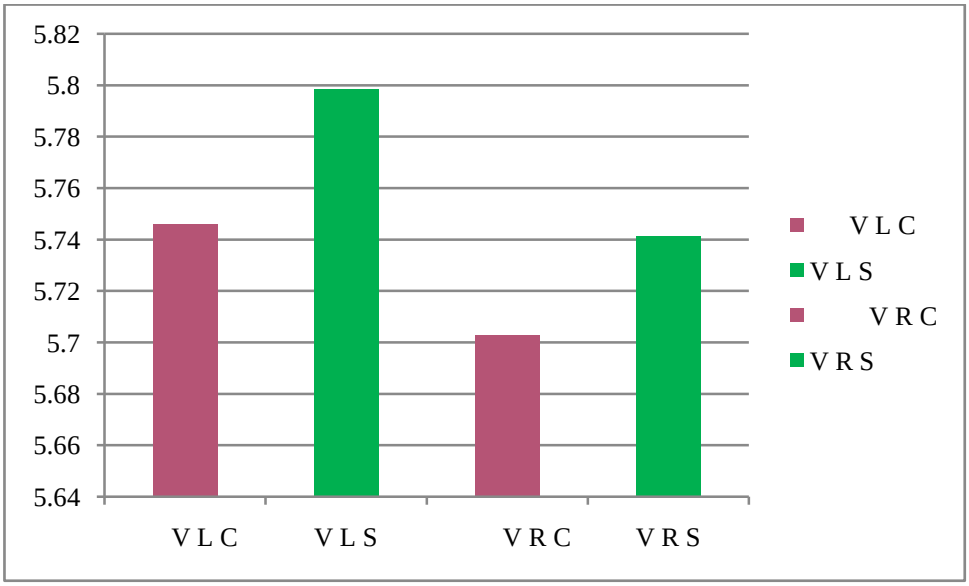

FIGURE:12

The Absolute Latency of Wave V of left side in VB TIA patients show the mean value of 5.7985±0.114635 and in the control group it is 5.74575±0.133607 with a P value of 0.02 showing the difference significant.

The Absolute Latency of Wave V of right side in VB TIA patients show the mean value of 5.74125±0.13915 and in the control group it is 5.703±0.09962 with a P value of 0.04 showing the difference significant.

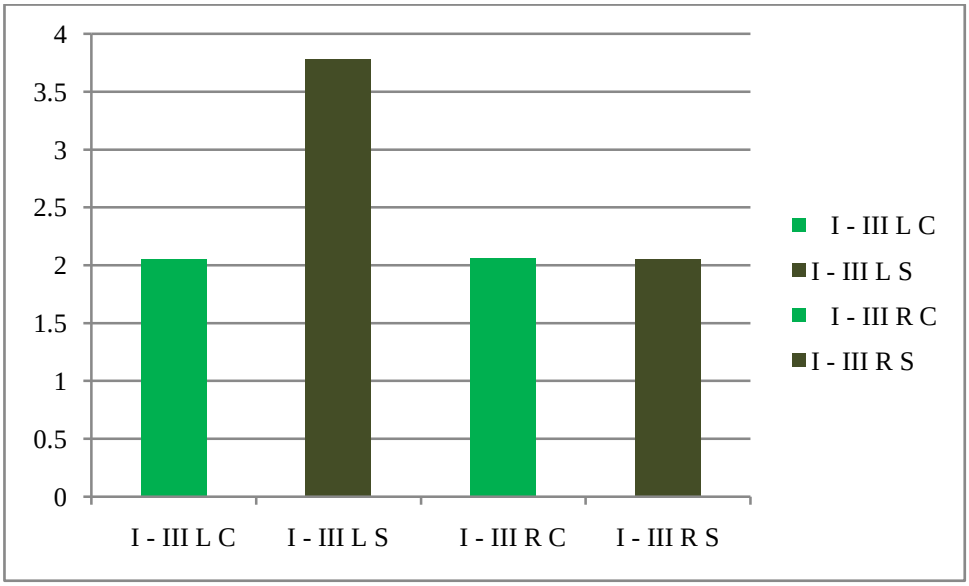

FIGURE:13

Interpeak latency Wave I-III of left side in VB TIA patients show the mean value of 2.06025 ±0.127192 and in the control group it is 2.05025 ±0.128731 with a P value of 0.44 showing the difference insignificant.

Interpeak latency Wave I-III of right side in VB TIA patients show the mean value of 2.04975 ± 0.11652 and in the control group it is 2.06025 ± 0.126054 with a P value of 0.25 showing the difference insignificant.

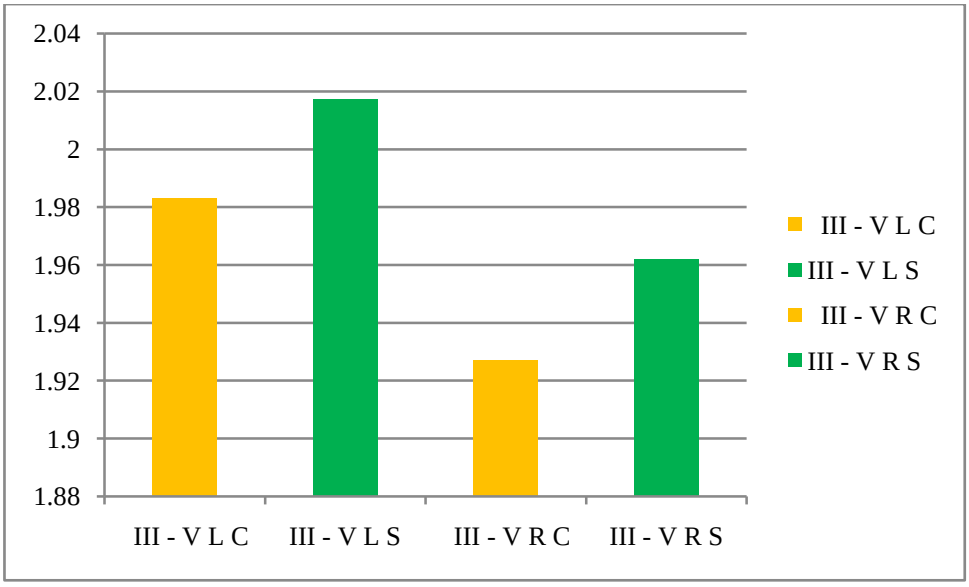

FIGURE:14

Interpeak latency Wave III - V of left side in VB TIA patients show the mean value of 2.01725 ± 0.162969 and in the control group it is 1.983±0.173104 with a P value of 0.13 showing the difference insignificant.

Interpeak latency Wave III - V of right side in VB TIA patients show the mean value of 1.962 ± 0.187195 and in the control group it is 1.927±0.154691 with a P value of 0.11 showing the difference insignificant

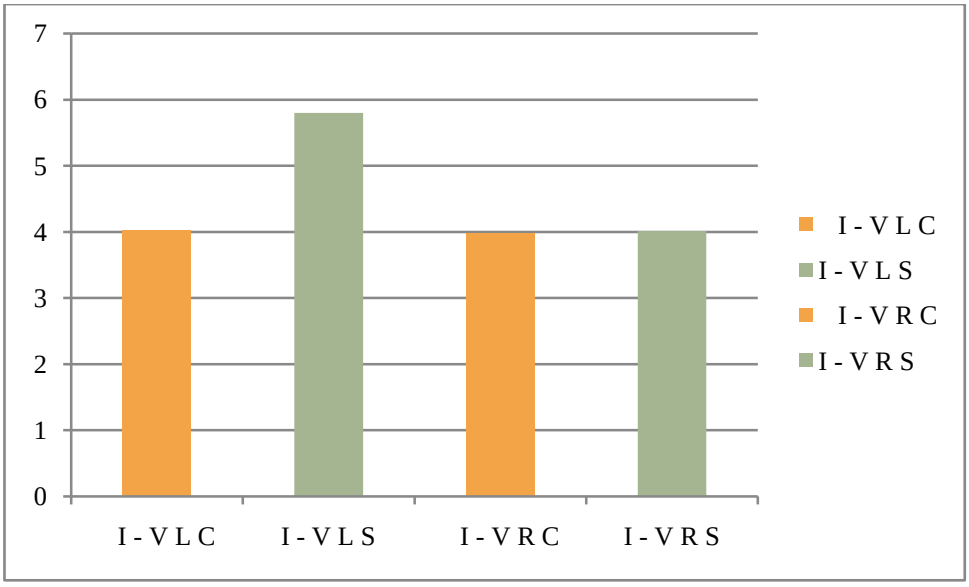

FIGURE:15

Interpeak latency Wave I-V of left side in VB TIA patients show the mean value of 4.071 ± 0.118013 and in the control group it is 4.03325 ± 0.157046 with a P value of 0.09 showing the difference insignificant.

Interpeak latency Wave I-V of right side in VB TIA patients show the mean value of 4.01175 ± 0.137204 and in the control group it is 3.98725 ± 0.106962 with a P value of 0.16 showing the difference insignificant

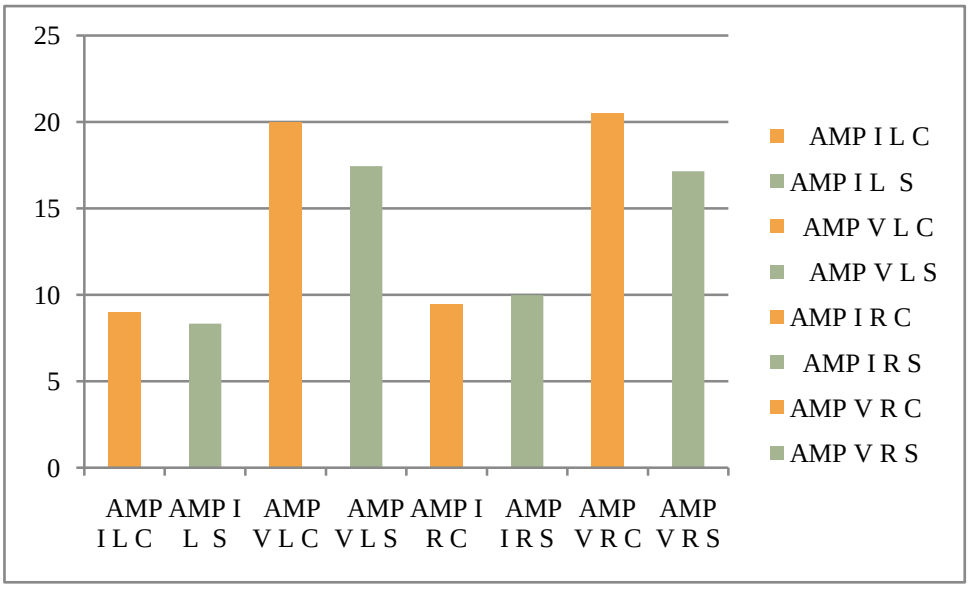

FIGURE:16

Amplitude of Wave I of left side in VB TIA patients show the mean value of 8.3375±2.630 and in the control group it is 8.978±3.619 with a P value of 0.19 showing the difference insignificant.

Amplitude of Wave I of right side in VB TIA patients show the mean value of 9.972±5.401 and in the control group it is 9.482±3.61 with a P value of 0.32 showing the difference insignificant.

Amplitude of Wave V of left side in VB TIA patients show the mean value of 17.449±6.882 and in the control group it is 19.95±5.67 with a P value of 0.04 showing the difference significant.

Amplitude of Wave V of right side in VB TIA patients show the mean value of 17.146±6.971 and in the control group it is 20.498±9.359 with a P value of 0.05 showing the difference significant.

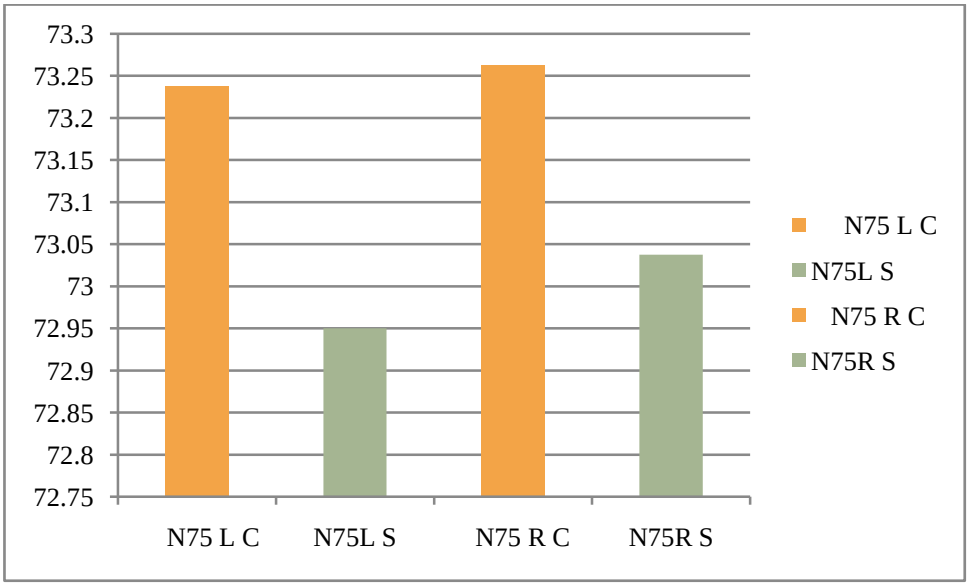

FIGURE:17

VISUAL EVOKED POTENTIALS:

The Absolute Latency of N 75 of left side in VB TIA patients show the mean value of 72.95±2.0933 and in the control group it is 73.2375± 2.345174 with a P value of 0.14 showing the difference insignificant.

The Absolute Latency of N 75 of left side in VB TIA patients show the mean value of 73.037±1.677 and in the control group it is 73.2625±2.081628 with a P value of 0.21 showing the difference insignificant.

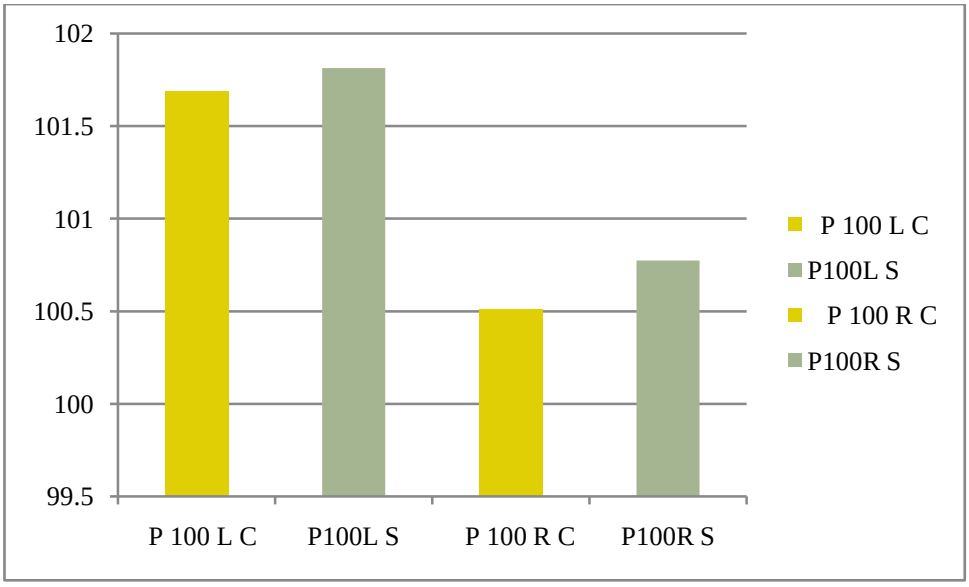

FIGURE:18

VISUAL EVOKED POTENTIALS:

The Absolute Latency of P 100 of left side in VB TIA patients show the mean value of 101.812 ± 3.553 and in the control group it is 101.687±4.8047 with a P value of 0.38 showing the difference insignificant.

The Absolute Latency of P 100 of Right side in VB TIA patients show the mean value of 100.775±1.822 and in the control group it is 100.512±2.8227 with a P value of 0.22 showing the difference insignificant

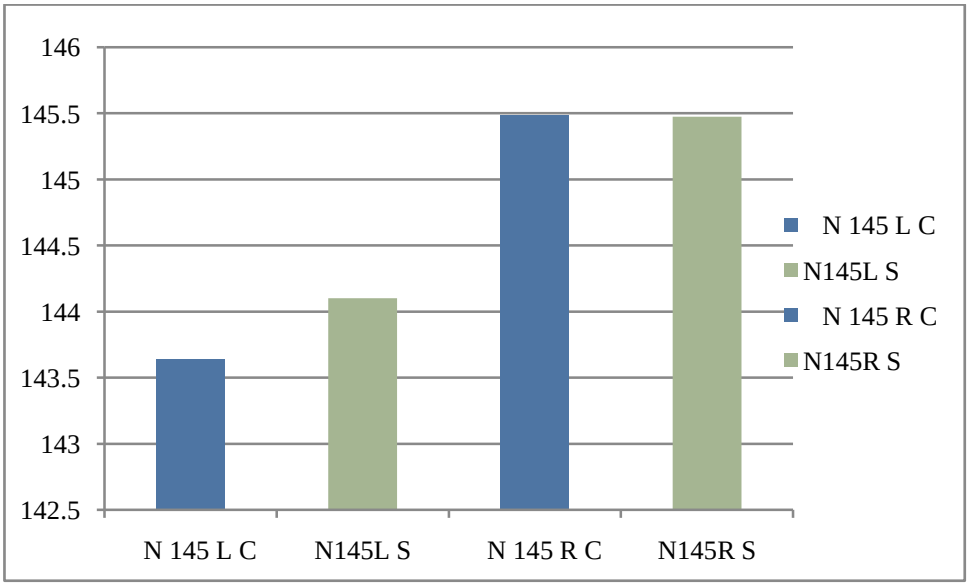

FIGURE:19

VISUAL EVOKED POTENTIALS:

The Absolute Latency of N 145 of left side in VB TIA patients show the mean value of 144.1± 2.539 and in the control group it is 143.637±3.2679 with a P value of 0.07 showing the difference insignificant

The Absolute Latency of N 145 of Right side in VB TIA patients show the mean value of 145.475 ± 2.832 and in the control group it is 145.487± 2.9515 with a P value of 0.44 showing the difference insignificant

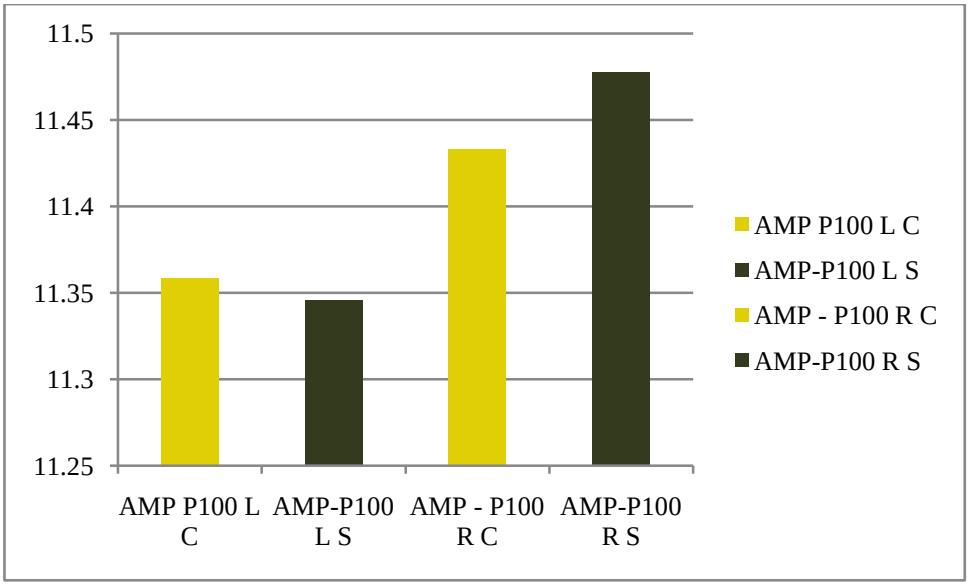

FIGURE:20

VISUAL EVOKED POTENTIALS:

Amplitude of P 100 of left side in VB TIA patients show the mean value of 11.345 ± 2.142257 and in the control group it is 11.358±2.221044 with a P value of 0.19 showing the difference insignificant.

Amplitude of P 100 of right side in VB TIA patients show the mean value of 11.477 ± 1.834364 and in the control group it is 11.433± 1.919383 with a P value of 0.32 showing the difference insignificant.

DISCUSSION:

In this study, the electrophysiological parameters were evaluated in patients with VB TIA. The results of the electrophysiological study were compared between 40 patients with VB TIA and 40 healthy subjects.

In this study, the Absolute Latency of Wave V is prolonged and reduction in Amplitude of Wave V in patients with VB TIA and is found to be statistically significant. It suggested that the diminished blood flow and alteration in cerebral metabolism during the course of transient ischemic attack may be the possible explanation for the amplitude reduction of BAEP. The amplitude depends on the intactness of the blood flow which is essential for the function of the brain.

In this study, no changes were found in the Interpeak latency of brainstem auditory evoked potential I-III,III-V ,and I-V in patients with VB TIA and statistically it was not significant.

This study showed that the symptoms relating to the VB TIA may be due to the reduction in blood flow to the brain especially the cerebellar cortex.

In the present study , empathized with the study of Benna P et al showed no changes were observed in the VEP parameters N75, P100 & N145.Similar Results were observed by Benna P et al. [5]

Stroke is an important consequence of VB TIA . TIA is a chronic process , in this

disease there will be a formation of atherosclerotic plaque. The symptoms are mainly due to the formation of emboli which may originate from other systemic organs. The resolution of symptom is due to collateral formation or lysis of emboli.

However the clinical observation and neuroimaging suggested that TIA had a deleterious effect and it will further lead to the development of stroke. Since the electrophysiological studies are useful to identify the changes in VB TIA patients, we could prevent them to develop the full blown stroke.

Measurement of stimulus conduction by means of evoked potentials is sensitive and reliable in the earlier detection of subclinical changes.

This study results are consistent with DRAKE ME et al, PENNA B et al showed significant reduction in amplitude of wave V and normal parameters of VEP suggested that there has been a reduction in blood flow in posterior circulation. In this study, there was a reduction in amplitude of wave V and Prolongation of Absolute Latency of Wave V of BERA in patients with VB TIA and it was statistically significant (P Value < 0.05) . These patients had a subclinical changes in vessel wall so that the blood flow was reduced. [4,5]

Mills JA et al evaluated brainstem auditory evoked potential in patients of reduced cerebral blood circulation and found that a significant change in absolute latency of wave V(P <0.05). and it was concluded that there is an alteration in myelination. [37]

Rossi L et al evaluated brainstem auditory evoked potential in VB TIA patients. They found a significant change in prolongation in Wave V latency ($P < 0.05$) and it was concluded that there is an alteration in myelination in VB TIA patients. [41]

CONCLUSION

Electrophysiological parameter like absolute latency of I,III,V and interpeak latencies of I-III, III-V, I-V , amplitude of wave I & V were evaluated. In the present Study , Amplitude reduction in Wave V and prolongation of Latency of Wave V of BERA were observed.

In VEP, No changes were observed in Latency and Amplitude. This study is very much useful to identify the early detection of progression of stroke in VB TIA Patients.

Finally further studies are required to evaluate the correlation between the electrophysiological parameters & progression into stroke . So that preventive measures can be used to prevent early involvement of Stroke.

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ANNEXURE

ABBREVIATIONS USED IN THE STUDY

TIA – TRANSIENT ISCHEMIC ATTACK

VB TIA – VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACK

BAEP -- BRAINSTEM AUDITORY EVOKED POTENTIAL

VEP – VISUAL EVOKED POTENTIAL

PCA –POSTERIAR CEREBRAL ARTERY

CSF – CEREBROSPINAL FLUID

HDL – HIGH DENSITY LIPOPROTEIN

LDL – LOW DENSITY LIPOPROTEIN

CBF – CEREBRAL BLOOD FLOW

MGN – MEDIAL GENICULATE NUCLEUS

INFORMED CONSENT FORM

Dr.R.Mohan, Post graduate student in the Department of physiology, Thanjavur Medical college, Thanjavur is studying the "**CHANGES OF AUDITORY EVOKED POTENTIAL AND VISUAL EVOKED POTENTIAL IN VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACKS PATIENTS "**

I understand the procedure and voluntarily agree to participate in the study, I also understand that this study is a non-invasive procedure and the possible adverse effects have been explained to me in details clearly in my own language.

Signature of the subject

Name:

Place:

Date:

PROFORMA

TOPIC: "CHANGES OF AUDITORY EVOKED POTENTIAL AND VISUAL EVOKED POTENTIAL IN VERTEBROBASILAR TRANSIENT ISCHEMIC ATTACKS PATIENTS"

Study group/Control group

Name: Age: Sex:

Address: Occupation:

H/O of Presenting illness:

H/o Giddiness

Past history: Known case of DM/SHT/PULMONARY TB/ASTHMA

Personal history: Smoking/alcohol/betalnut chewing with or without tobacco

General Examination:

Height: Weight

Anaemic / Not anaemic

Cyanosis / No cyanosis

Clubbing / No Clubbing

Jaundice / No jaundice

Pedal oedema / No pedal oedema

Generalised lymphadenopathy present/Absent

Vital Signs: PR: BP: RR:

Examination of CVS:

Examination of RS:

Examination of Abdomen:

Examination of CNS:

Investigations:

BRAINSTEM AUDITORY EVOKED RESPONSE

WAVE LATENCY (ms)	I	III	V	I-III IPL	III-V IPL	I-V IPL
RIGHT						
LEFT						

AMPLITUDE (μV)	I	V
RIGHT		
LEFT		

VISUAL EVOKED POTENTIAL RESPONSE

LATENCY(ms)	N 75	P 100	N 145
RIGHT			
LEFT			

AMPLITUDE P 100 (µV)	
RIGHT EYE	
LEFT EYE	

MASTER CHART

	BERA LATENCYCONTROL (ms)													BERA AMPLITUDE (µV)			
S. NO	AGE / SEX	I L C	I R C	III L C	III R C	V L C	V R C	I - III L C	I - III R C	III - V L C	III - V R C	I - V L C	I - V R C	AMP I L C	AMP V L C	AMP I R C	AMP V R C
1	55/F	1.68	1.65	3.7	3.65	5.5	5.6	2.02	2	1.8	1.95	3.82	3.95	11.21	16.52	7.24	15.31
2	56/M	1.7	1.65	3.85	3.82	5.68	5.42	2.15	2.17	1.83	1.6	3.98	3.77	5.31	18.51	6.34	32.12
3	54/M	1.7	1.65	3.68	3.88	5.72	5.68	1.98	2.23	2.04	1.8	4.02	4.03	8.12	24.31	14.51	8.43
4	67/M	1.72	1.72	3.72	3.92	5.78	5.72	2	2.2	2.06	1.8	4.06	4	9.34	15.32	15.28	24.15
5	62/F	1.62	1.72	3.62	3.78	5.8	5.72	2	2.06	2.18	1.94	4.18	4	10.15	9.15	7.34	9.61
6	64/M	1.7	1.72	3.9	3.78	5.6	5.78	2.2	2.06	1.7	2	3.9	4.06	8.64	12.36	6.21	27.02
7	62/M	1.65	1.7	3.72	3.85	5.78	5.65	2.07	2.15	2.06	1.8	4.13	3.95	7.15	22.35	8.31	16.05
8	56/F	1.62	1.65	3.75	3.88	5.72	5.7	2.13	2.23	1.97	1.82	4.1	4.05	7.75	15.42	15.64	27.46
9	58/M	1.68	1.68	3.68	3.95	5.56	5.6	2	2.27	1.88	1.65	3.88	3.92	14.13	25.34	14.96	12.01
10	58/M	1.75	1.65	3.9	3.75	5.6	5.7	2.15	2.1	1.7	1.95	3.85	4.05	5.31	17.32	8.12	9.12
11	54/M	1.8	1.72	3.82	3.62	5.75	5.82	2.02	1.9	1.93	2.2	3.95	4.1	6.16	23.05	15.21	35.08
12	52/F	1.72	1.82	3.72	3.82	5.88	5.7	2	2	2.16	1.88	4.16	3.88	9.23	24.15	15.62	15.15
13	51/F	1.65	1.65	3.75	3.78	5.7	5.48	2.1	2.13	1.95	1.7	4.05	3.83	9.12	18.43	15.36	32.15
14	56/M	1.82	1.75	3.65	3.72	5.65	5.7	1.83	1.97	2	1.98	3.83	3.95	9.52	25.56	8.75	18.31
15	68/F	1.75	1.7	3.75	3.75	5.88	5.68	2	2.05	2.13	1.93	4.13	3.98	11.25	8.75	8.87	8.16
16	62/F	1.82	1.62	3.72	3.8	5.6	5.82	1.9	2.18	1.88	2.02	3.78	4.2	9.72	22.15	5.64	27.16
17	60/M	1.78	1.75	3.72	3.7	5.7	5.78	1.94	1.95	1.98	2.08	3.92	4.03	8.35	20.45	3.31	15.01
18	57/F	1.65	1.78	3.65	3.65	5.82	5.78	2	1.87	2.17	2.13	4.17	4	9.65	15.62	5.57	8.32
19	54/F	1.78	1.62	3.8	3.62	5.78	5.7	2.02	2	1.98	2.08	4	4.08	4.87	13.48	7.34	20.05
20	53/M	1.68	1.65	3.98	3.7	5.95	5.78	2.3	2.05	1.97	2.08	4.27	4.13	8.43	15.38	9.02	22.86

S. NO	AGE / SEX	I L C	I R C	III L C	III R C	V L C	V R C	I - III L C	I - III R C	III - V L C	III - V R C	I - V L C	I - V R C	AMP I L C	AMP V L C	AMP I R C	AMP V R C
21	57/F	1.78	1.72	3.68	3.85	5.6	5.6	1.9	2.13	1.92	1.75	3.82	3.88	14.15	18.54	5.45	32.12
22	62/M	1.72	1.65	3.78	3.85	5.82	5.85	2.06	2.2	2.04	2	4.1	4.2	5.67	27.32	8.06	35.21
23	64/F	1.7	1.78	3.8	3.78	5.78	5.8	2.1	2	1.98	2.02	4.08	4.02	5.32	18.43	8.45	17.36
24	63/M	1.68	1.7	3.78	3.68	5.42	5.78	2.1	1.98	1.64	2.1	3.74	4.08	4.76	24.51	10.13	34.42
25	64/F	1.65	1.75	3.78	3.75	5.75	5.75	2.13	2	1.97	2	4.1	4	5.16	36.58	7.34	42.31
26	56/M	1.8	1.9	3.68	3.75	5.75	5.7	1.88	1.85	2.07	1.95	3.95	3.8	13.32	28.47	8.03	14.52
27	54/M	1.75	1.85	3.78	3.75	5.7	5.78	2.03	1.9	1.92	2.03	3.95	3.93	4.12	21.42	6.31	25.61
28	58/M	1.78	1.7	3.85	3.55	5.72	5.8	2.07	1.85	1.87	2.25	3.94	4.1	8.57	18.53	5.24	16.21
29	67/F	1.65	1.75	3.78	3.7	5.85	5.72	2.13	1.95	2.07	2.02	4.2	3.97	7.32	22.27	4.58	23.24
30	62/M	1.78	1.62	3.88	4.08	5.45	5.7	2.1	2.46	1.57	1.62	3.67	4.08	16.24	17.42	12.13	12.01
31	61/F	1.75	1.62	3.75	3.75	6.02	5.75	2	2.13	2.27	2	4.27	4.13	12.54	19.31	15.62	9.61
32	64/M	1.85	1.8	3.45	3.9	5.95	5.78	1.6	2.1	2.5	1.88	4.1	3.98	5.21	16.51	9.43	7.05
33	61/F	1.6	1.8	3.78	3.9	5.85	5.8	2.18	2.1	2.07	1.9	4.25	4	15.24	23.54	13.23	25.43
34	59/F	1.7	1.8	3.9	3.78	5.8	5.7	2.2	1.98	1.9	1.92	4.1	3.9	18.26	18.54	10.08	16.54
35	60/M	1.62	1.85	3.68	3.85	5.88	5.8	2.06	2	2.2	1.95	4.26	3.95	7.53	24.51	12.13	8.35
36	64/M	1.7	1.72	3.68	3.8	5.8	5.65	1.98	2.08	2.12	1.85	4.1	3.93	5.72	17.45	8.57	14.53
37	58/M	1.75	1.8	3.75	3.75	5.72	5.7	2	1.95	1.97	1.95	3.97	3.9	6.42	17.45	5.61	25.24
38	57/M	1.65	1.6	3.85	3.58	5.92	5.6	2.2	1.98	2.07	2.02	4.27	4	15.12	16.31	12.31	18.75
39	52/F	1.62	1.75	3.8	3.85	5.75	5.45	2.18	2.1	1.95	1.6	4.13	3.7	4.51	15.12	9.43	25.75
40	57/M	1.7	1.62	4	3.72	5.85	5.6	2.3	2.1	1.85	1.88	4.15	3.98	10.54	32.15	8.54	32.13

VEP LATENCY (ms)						VEP AMPLITUDE (μv)	
CONTROL						CONTROL	
N75 L	N75 R	P 100 L	P 100 R	N 145 L	N 145 R	AMP - P100 L	AMP - P100 R
74	75	97	95	145	142.5	10.32	13.8
76.5	77	91.5	100	138	146	12.58	13.75
70.5	71	101	100	146	145	12.89	12.72
71.5	74	94.5	101	138	146.5	13.65	13.87
71	74.5	101	95	145	146	11.72	12.65
71.5	70.5	101.5	102.5	138.5	145.5	13.21	13.56
72.5	75	104.5	106	146	148.5	7.48	12.83
72.5	73	99.5	104.5	146	148.5	12.47	7.65
76.5	72.5	101.5	103	147	149	8.36	8.52
74.5	74.5	100.5	102.5	138.5	144	13.45	9.52
70.5	74	99.5	100.5	145	152	9.46	11.34
73	71	103.5	102	145	148.5	12.84	12.67
75.5	75.5	100	100.5	141	145.5	13.83	8.32
76.5	76.5	102	101	138	147	9.85	9.76
73	72.5	99.5	105.5	146.5	144	14.23	14.02
72.5	73	105.5	99.5	145.5	145	12.64	12.37
71.5	73.5	98.5	98	148	142	14.06	12.42
70	73.5	103.5	102	141	145.5	13.5	9.42
70.5	73	99	98	144	151.5	12.01	10.46
78.5	73	100.5	101	151	141.5	7.01	12.54

70.5	73.5	105	101	144	147.5	8.01	11.37
70	72.5	101.5	101	142.5	147.5	9.28	10.14
73.5	71	101	100	146	146.5	9.6	8.5
73	71.5	101	100	144	144.5	12.97	11.92
71.5	71	103.5	100	146	145	9.51	10.18
80	72.5	115.5	100	138.5	145	7.7	8.2
73	77	97.5	98	143	145	11.56	12.58
73.5	70	98	102.5	144	141.5	8.44	14.02
73.5	75	106.5	104.5	142.5	144	13.22	11.49
74.5	74	102	100	144	144.5	12.37	12.67
75	75.5	117.5	99.5	146.5	141.5	13.2	13.28
74.5	74	108.5	108	138	147	12.18	12.11
73.5	73.5	102	98	142.5	139	13.14	9.58
69.5	70.5	97.5	101.5	148	144	8.23	13
75	78.5	96.5	98	144	141	12.31	13.18
74.5	69	103	96.5	143	148	12.96	8.58
74	71	99.5	102	148.5	146	13.05	9.56
72	73.5	102	97	144	148	13.15	12.08
71.5	73	98	98.5	141	140	10.45	12.92
74.5	71.5	107.5	97	142.5	150	7.45	9.78

BERA LATENCY SUBJECT(ms)														BERA AMPLITUDE SUBJECT(µV)			
S.NO	AGE / SEX	I L	I R	III L	III R	V L	V R	I - III L	I - III R	III - V L	III - V R	I - V L	I - V R	I L	V L	I R	V R
1	55/M	1.78	1.62	3.82	3.72	5.85	5.68	2.04	2.1	2.03	1.96	4.07	4.06	7.26	9.35	12.13	15.64
2	64/F	1.62	1.76	3.88	3.88	5.82	5.52	2.26	2.12	1.94	1.64	4.2	3.76	5.28	12.53	8.26	23.52
3	56/M	1.72	1.72	3.65	4.02	5.92	5.78	1.93	2.3	2.27	1.76	4.2	4.06	8.26	13.54	10.15	15.34
4	63/M	1.7	1.74	3.85	3.88	5.92	5.85	2.15	2.14	2.07	1.97	4.22	4.11	4.24	25.34	5.13	25.06
5	66/M	1.62	1.68	3.65	3.62	5.72	5.78	2.03	1.94	2.07	2.16	4.1	4.1	6.21	9.21	12.23	19.35
6	62/F	1.62	1.68	3.82	3.88	5.52	5.55	2.2	2.2	1.7	1.67	3.9	3.87	10.42	15.21	6.54	22.01
7	59/M	1.78	1.67	3.68	3.78	5.98	5.52	1.9	2.11	2.3	1.74	4.2	3.85	12.51	25.31	8.32	8.31
8	57/F	1.72	1.7	3.72	3.82	5.55	5.75	2	2.12	1.83	1.93	3.83	4.05	8.71	26.34	15.34	8.62
9	55/F	1.75	1.72	3.68	3.92	5.78	5.65	1.93	2.2	2.1	1.73	4.03	3.93	9.34	15.61	7.18	11.42
10	56/M	1.62	1.76	3.92	3.72	5.7	5.75	2.3	1.96	1.78	2.03	4.08	3.99	8.36	18.31	3.26	13.52
11	58/M	1.7	1.87	3.92	3.68	5.58	5.74	2.22	1.81	1.66	2.06	3.88	3.87	10.51	12.61	5.54	9.65
12	54/F	1.82	1.76	3.78	3.8	5.62	5.75	1.96	2.04	1.84	1.95	3.8	3.99	13.42	8.61	12.21	15.34
13	56/M	1.7	1.69	3.68	3.82	5.92	5.42	1.98	2.13	2.24	1.6	4.22	3.73	8.36	14.51	6.12	9.34
14	64/F	1.78	1.78	3.78	3.75	5.62	5.8	2	1.97	1.84	2.05	3.84	4.02	7.34	8.31	4.15	15.16
15	63/M	1.75	1.82	3.7	3.68	5.92	6.02	1.95	1.86	2.22	2.34	4.17	4.2	3.58	14.61	15.42	18.26
16	62/M	1.8	1.76	3.98	4.02	5.72	5.6	2.18	2.26	1.74	1.58	3.92	3.84	5.21	26.35	12.51	14.52
17	59/F	1.68	1.8	3.65	3.65	5.78	5.72	1.97	1.85	2.13	2.07	4.1	3.92	7.34	18.54	7.45	11.31
18	54/M	1.72	1.7	3.78	3.6	5.88	5.65	2.06	1.9	2.1	2.05	4.16	3.95	5.87	15.61	5.62	12.62
19	55/M	1.65	1.65	3.72	3.7	5.8	5.58	2.07	2.05	2.08	1.88	4.15	3.93	5.24	12.34	5.31	25.21
20	59/F	1.62	1.72	3.88	3.65	5.75	5.82	2.26	1.93	1.87	2.17	4.13	4.1	12.31	15.61	12.32	8.34

21	52/ F	1.72	1.68	3.72	3.78	5.85	5.78	2	2.1	2.13	2	4.13	4.1	5.34	8.28	15.32	22.52
22	57/ M	1.75	1.64	3.82	3.78	5.7	5.75	2.07	2.14	1.88	1.97	395	4.11	9.65	14.51	10.15	9.35
23	69/ F	1.62	1.72	3.75	3.82	5.8	5.82	2.13	2.1	2.05	2	4.18	4.1	5.34	12.31	8.56	27.56
24	61/M	1.65	1.65	3.65	3.7	5.72	5.72	2	2.05	2.07	2.02	4.07	4.07	11.21	8.58	14.51	19.68
25	67/M	1.78	1.69	3.75	3.68	5.92	6.08	1.97	1.99	2.17	2.4	4.14	4.39	9.65	15.37	3.25	8.01
26	62/F	1.72	1.82	3.82	3.8	5.82	5.82	2.1	1.98	2	2.02	4.1	4	12.34	15.61	22.15	13.45
27	53/M	1.68	1.79	3.78	3.82	5.85	5.72	2.1	2.03	2.07	1.9	4.17	3.93	12.23	18.34	6.45	28.35
28	52/M	1.88	1.76	3.82	3.65	5.72	5.65	1.94	1.89	1.9	2	3.84	3.89	7.46	18.57	14.45	16.35
29	57/F	1.62	1.64	3.85	3.82	5.82	5.78	2.23	2.18	1.97	1.96	4.2	4.14	6.78	20.15	8.75	9.35
30	58/M	1.72	1.75	3.92	3.98	5.8	5.72	2.2	2.23	1.88	1.74	4.08	3.97	6.31	12.54	19.02	21.03
31	60/M	1.62	1.7	3.95	3.75	5.72	5.62	2.33	2.05	1.77	1.87	4.1	3.92	12.57	20.51	3.02	27.35
32	60/F	1.7	1.72	3.62	3.88	5.78	5.72	1.92	2.16	2.16	1.84	4.08	4	8.63	24.57	21.24	16.34
33	61/F	1.82	1.78	3.75	3.88	5.88	6.05	1.93	2.1	2.13	2.17	4.06	4.27	8.35	16.95	9.16	14.65
34	55/M	1.85	1.72	3.92	3.75	5.92	5.98	2.07	2.03	2	2.23	4.07	4.26	7.75	18.54	16.21	31.64
35	54/M	1.78	1.76	3.65	3.82	5.78	5.82	1.87	2.06	2.13	2	4	4.06	5.89	18.42	5.46	14.53
36	53/F	1.8	1.68	3.65	3.75	5.88	5.65	1.85	2.07	2.23	1.9	4.08	3.97	6.73	32.41	3.14	30.05
37	61/M	1.75	1.75	3.78	3.72	5.92	5.72	2.03	1.97	2.14	2	4.17	3.97	10.65	12.65	21.36	14.51
38	59/M	1.82	1.75	3.78	3.65	5.88	5.85	1.96	1.9	2.1	2.2	4.06	4.1	8.12	26.43	12.54	31.23
39	57/M	1.85	1.83	3.75	3.8	5.85	5.65	1.9	1.97	2.1	1.85	4	3.82	6.53	37.84	5.94	12.15
40	64/F	1.82	1.75	3.98	3.75	5.98	5.82	2.16	2	2	2.07	4.16	4.07	12.21	27.56	3.02	15.21

VEP LATENCY (ms)						VEP AMPLITUDE (μV)	
SUBJECT						SUBJECT	
N75L	N75R	P100L	P100R	N145L	N145R	AMP-P100L	AMP-P100R
75.5	74.5	99.5	97.5	145.5	142	11.32	13.58
75	75	95	99.5	145	145.5	12.64	13.5
69.5	72.5	96.5	97	145.5	145	13.02	12.5
71	75.5	98.5	102	137.5	147	13.45	13.5
71.5	72.5	99.5	100.5	145	145	12.05	12.5
72	72.5	100	100	145	145	13.45	13.84
74	74	102.5	104.5	145.5	147.5	7.58	13
74	72.5	102	102	146	148	11.87	8.02
75.5	70	103.5	102	145.5	148.5	8.4	8.34
71	72.5	99.5	100.5	144	144.5	12.85	9.46
72	72	102	99.5	145	152	9.2	11.5
71	70.5	102	102.5	145.5	148	12.5	12.5
77	74	103.5	99.5	141.5	145	13.5	8.5
75	75	103.5	103	142	147.5	9.5	9.5
72.5	70.5	102	102	145.5	144	14	13.85
70	71	102.5	100.5	145	145	12.5	12.5
70	71.5	100	99.5	147.5	143	13.89	12.05
72	74	100	101.5	141	145	13.45	9.84
73.5	72.5	100.5	99.5	144.5	151	12.45	10.5
75.5	75.5	102	102.5	149.5	141	7.05	12.58

69.5	75	104.5	102.5	143.5	148	8.05	11.45
72.5	73.5	100	98.5	142.5	147.5	9.5	10.35
72	70.5	102.5	99	145.5	146.5	9.5	9
71.5	72.5	100	101.5	145	145	12.5	12.05
73	73	102.5	102	145.5	145	9.45	10.42
78	74.5	107.5	102.5	138.5	145	7.5	8.45
74.5	74.5	102.5	100	142.5	144.5	11.26	12.85
75	72.5	100	103	145	141	8.5	13.85
72	72.5	105	101.5	145	145.5	13.02	12
72.5	72	100.5	98.5	144.5	145	12.5	12.5
74.5	77	115	97	145.5	142	13	13.2
74	72.5	110.5	105	138.5	147.5	12.45	12.5
75	71.5	100	100.5	141.5	138.5	13.25	9.75
70	72	100	102	148	145	8.5	13.05
72.5	75	98.5	100.5	143.5	142	12.5	13.5
73	70.5	103.5	100	143	148.5	12.5	8.45
72.5	73	97.5	101	148	145.5	13.1	9.75
70	75	103.5	100	143.5	147.5	13.2	11.85
73.5	74.5	99.5	101	141.5	140.5	11.02	12.76
75	72	105	99.5	142	149.5	7.85	9.82